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COMMUNICATIONS

A CONTRIBUTION TO THE PATHOLOGY OF BOWMAN'S MEMBRANE*

BY

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THE function of the ocular glass membranes is not fully understood. The two elastic membranes, Descemet's and Bruch's, may contribute to the firmness of the globe and counterbalance the varying intra-ocular pressure. Besides this essential function they may control the metabolism of tissues linked with them. The cornea, lens, and retina demand a very constant constitution of the lymph-stream. These glass membranes may fulfil a special filter function. It may be that observation of pathological changes of these structures may contribute to the knowledge of their function.

Thirty years ago Ernst Fuchs (*Arch. f. Ophthal.*, Vol. LXXXIX, p. 337) described a case of familial corneal dystrophy, in which Bowman's membrane divided the corneal incrustations into two layers. That in front of Bowman's membrane was acido-(eosino) phil, the granules posterior to Bowman's membrane being Giemsa blue and basophil. The acidophil substance was flat and structureless, while the blueish basophil granules of varying size and shape were prised between the corneal lamellae (Loewenstein, *Amer. Jl. Ophthal.*, Vol. XXIII, p. 1,229, 1940). The two kinds of

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incrustation are separated according to their pH concentration by a membrane. This is an unusual occurrence in histological studies. Another peculiarity of the ocular glass membranes is the fact that they undergo a change due to age, for example, the appearance of Henle's warts, drusen of Bruch's membrane and the fatty impairment of Bowman's membrane in arcus senilis. Wear and tear of life seems to damage glass membranes at an earlier term than the surrounding tissues. A metabolism inferior to that of vascularised tissues is likely in glass membranes.

I.—Band-shaped corneal degeneration

Metabolic changes in the eye may lead to the gross change in Bowman's membrane known as band-shaped corneal degeneration. Physical and chemical conditions basic to this condition are unknown.

The specimen which we wish to discuss was sent by Dr. Magnus of York. A married woman, aged 67 years, suffered from uveitis for several years. There was no history of infantile rheumatism. General investigations were negative except for a strongly positive Mantoux reaction. A course of tuberculin treatment was given. In 1942 a bilateral iridectomy was carried out. Broad prominent band-shaped incrustations with numerous tears and clefts were removed from the left eye in January, 1945. They had the appearance of broken ice. A conjunctival bridge flap was used to cover the defect. A similar procedure was carried out in the right eye without a conjunctival flap. Both eyes settled down without reaction. Vision R. and L. 6/60. Three pieces of the specimen were sent to me; two were embedded in paraffin and the third in gelatine. Fat staining proved to be negative. The paraffin slides were stained with haematoxylin and eosin, van Gieson, Mallory, Masson and von Kossa (for lime).

In front of Bowman's membrane there was eosin-staining tissue which appeared to be whorled. The eosined mass was loose in some areas and compact with empty holes at others. Where the epithelium had gone, the acidophil mass covered the front of Bowman's membrane (Fig. 1) and was three or four times the normal thickness of epithelium. The red areas were the shape of onion peelings lying between preserved corneal epithelium (Fig. 2). At some places the acidophil mass was poised as a flat structureless smooth layer in front of Bowman's membrane (Fig. 3) and was reminiscent of the findings in familial corneal dystrophy. There was an indistinct, slightly red mass with few nuclei at some places subepithelially (Fig. 1), at others a connective tissue was found (Fig. 4) not unlike that found in old degenerative pannus. Bowman's membrane was calcified throughout the whole specimen.

No gas bubbles arose on adding 50 per cent. HCl, but the substance of Bowman's membrane was melted away to a considerable extent. Von Kossa's reaction was strongly positive and therefore a Ca compound was present; this may have been calcium phosphate (Fig. 5).

Not all the granules in Bowman's membrane and the neighbouring lamellae were brownish black with von Kossa's reagent as some granules appeared safranin red. We may conclude that calcification is a later stage of the regressive process. The safranin stained granules may be a protein precipitate, a possible forerunner of calcification. Bowman's membrane appeared equally granular and stained a dark purple with haematoxylin; at other places it was split into thin platelets like mica (Fig. 6). The anterior lamellae were the thickest and darkest. Elsewhere the whole thickness of Bowman's membrane was broken into smaller fragments. The preserved epithelium grew between the gaps into the corneal lamellae (Fig. 1). The unequal distribution of lime was shown at other places by very small and patchlike purple staining.

Splitting of Bowman's membrane in parallel platelets was due to pathological changes, and it is likely that we have to deal with a peculiarity which is based on a preformed physiological structure. According to Salzmann, Eisler, Wolff and others, there is no structure recognisable in Bowman's membrane. Its fibrils are supposed to be closely interwoven. Nevertheless the appearance of mica-like regular platelets in the calcified tissue is hard to explain otherwise than by physiological preformation. Bruch's membrane for instance appears in routine examination as an entity, but is split pathologically or can be split by special histological methods. In cases of lipoid infiltration Descemet's membrane is affected only in its anterior third (Loewenstein, *Trans. Ophthal. Soc., U.K.*, Vol. LXII, p. 159, 1942), the posterior two-thirds being fat free.

How can one explain the establishment of lime incrustation in Bowman's membrane with a scattering in the superficial lamellae? Fischer (*Arch. f. Augenheilk.*, Vol. CII, p. 146, 1929) has shown that cornea with preserved epithelium and endothelium is permeable to water from the conjunctival side. Oxygen can diffuse through the cornea towards the aqueous and CO_2 only outwards towards the air. It seems that a certain equilibrium of gas metabolism is necessary to keep Bowman's membrane intact. We know that calcium phosphate is precipitated if the pH concentration of the tissue fluid changes from the acid to the alkaline side. That may happen when CO_2 escapes. As the fibrils of Bowman's membrane are very closely interwoven, a minor exchange of fluid is more probable than in the looser tissue of the parenchyma, where lymph circulates under better conditions. We may assume that a

reduced metabolism leads to alkalosis of the corneal tissue. Calcium phosphate is precipitated from the corneal tissue fluid in the part of the cornea which has the slowest metabolism—that is in Bowman's membrane. It may be that the direction of the fluid stream causes the deposit in Bowman's membrane and not in Descemet's membrane.

It is easily understood that permeability of Bowman's membrane suffers by calcification and the fluid stream from the epithelial side towards the anterior chamber is impeded thus explaining precipitation in front of Bowman's membrane. We cannot offer an explanation why those precipitates are eosinophil. It is not blood which produces the acidophil mass, for in slides stained with Mallory or Masson, red blood corpuscles appear orange while the acidophil areas are an intensive red. The appearance of this difference in front and behind the frontal layer of Bowman's membrane in familial corneal dystrophy and in band-shaped corneal degeneration is remarkable.

We would expect the calcified Bowman's membrane to lose its elasticity, to break up in many places, and the epithelium might grow into the corneal lamellae. We conclude from the histological appearance that the abnormal position of the calcified broken ends is bound to create a chronic irritation. Improvement is possible by operative removal of the prominent sharp edges.

The question of lime precipitation in the cornea was discussed by Axenfeld, in his last paper: Dystrophies of the corneal parenchyma (*Klin. Monatsbl. f. Augenheilk.*, Vol. LXXXV, p. 493, 1930). The primary disease, according to Axenfeld, is located within the cement substance between the corneal lamellae. This substance may be impregnated with lipoidal bodies as in fatty, symmetrical, primary corneal dystrophy, or in corneal arcus lipoides. It may be an impregnation with hyaline or mucinous material or finally with lime, each of these substances causing a different kind of corneal dystrophy.

Axenfeld considers that light may produce precipitation of lime salts in the cornea. This idea may be especially productive in the explanation of the establishment of calcium salts in Bowman's membrane in cases of band-shaped corneal degeneration, as they are restricted to the part of the cornea, almost always, exposed to air and light. It is more than likely that a slowed down corneal metabolism, as it might occur, and is supposed to occur, in chronic iridocyclitis, may be prone to be influenced by light rays.

Finally I want to mention that I have observed two cases of young girls (aged 8 and 10 years) with chronic uveitis of both eyes and rheumatic affections of the lower extremities. Both showed a well developed icy band-shaped corneal degeneration in each eye. In spite of the softness of the painless unirritated eyes the vision

was still 6/36 and 6/24 respectively. Calcium was not increased in the blood. The band-shaped corneal degeneration in the lid fissure is assumed in these cases to be due to local metabolic deterioration. The influence of light may have facilitated the establishment of the precipitates in Bowman's membrane. Calcium opacities in Axenfeld's case of calcareous corneal dystrophy were influenced favourably by dense light filters worn for a long time (dark auto-goggles). It is worth while to try a similar therapy in those cases of calcareous deposits in Bowman's membrane. It might be tried in other cases of corneal dystrophies as well, as a similar modus of precipitation may come into consideration.

Our speculation is that there is a slowing down of the corneal metabolism in band-shaped corneal dystrophy which leads to alkalosis. Calcium phosphate is precipitated in Bowman's membrane. The calcified Bowman's membrane is a barrier to the transfer of fluid from the epithelium through the corneal tissue. Thus an eosinophil mass is established in front of Bowman's membrane. These greyish white masses are responsible for the broken ice appearance of band-shaped corneal degeneration. Calcified Bowman's membrane loses its elasticity and breaks up under the stress of wear and tear.

II.—Bowman's membrane in glaucomatous eyes

If this idea is correct, we may expect changes in Bowman's membrane whenever corneal metabolism is damaged by increased intra-ocular pressure. It is really astonishing that such a frequent appearance as the hemispherical bodies in Bowman's membrane in glaucomatous eyes were overlooked before E. von Hippel (Cornea in Wessely's Pathology of the Eye, Vol. 1, p. 322, Springer, 1928) described them without giving an explanation. E. Wolff and Lyle (*Proc.* Roy. Soc. Med., Vol. VI, p. 701, 1932 and Talbot, *Brit. Jl. Ophthal.*, Vol. XXII, p. 210, 1938) have confirmed their presence in glaucoma cases. We have seen these bodies very frequently (Fig. 7) in cases of primary and secondary glaucoma and also in hydrophthalmos. Garrow and Loewenstein (*Brit. Jl. Ophthal.*, Vol. XXV, p. 514, 1941) infer that they are an indication of disordered corneal metabolism in glaucomatous eyes.

The fluid stream towards the anterior chamber reaches the rigid scaffolding of the globe after passing the loose epithelial covering. We assume that an obstacle is created by the increased intra-ocular pressure and precipitation occurs where the fluid stream is checked, that is, at the outer, the epithelial side of Bowman's membrane, where the hemispherical bodies are found exclusively. The presence of hemispherical bodies has nothing to do with the cause of primary glaucoma, but is a corollary only of the increased intra-ocular pressure. It is found in different kinds of secondary glaucoma.

III.—Concretion in Bowman's membrane in a case of hypertensive retinopathy

We have found another kind of precipitation in Bowman's membrane in both eyes of a patient with hypertensive retinopathy, undescribed so far. These precipitates were discovered in the periphery whereas the glaucomatous hemispherical bodies show no predilection for any part of the cornea. This second type were not round, but ragged (Fig. 8) and many were polyhedric (Fig. 9). They are bigger and extend throughout a whole thickness of Bowman's membrane. Some of them find a continuation in the superficial corneal lamellae. Similar concretions were present in the basal layers of the epithelium of the sclera and in the fibres of the episcleral tissue (Fig. 10). We have not found any description of this kind of concretion so far. Elschnig's drusen of Bowman's membrane (*Klin. Monatsb. f. Augenheilk.*, Vol. XXXIII, p. 453, 1899) and Loewenstein's colloid droplets in the anterior corneal lamellae (*Klin. Monatsb. f. Augenheilk.*, Vol. L, p. 513, 1912) are different in appearance. This observation seems to prove that senile changes and other regressive processes linked with hypertensive retinopathy might create another disturbance of corneal metabolism which differs from that seen in glaucoma.

IV.—Bowman's membrane in a case of late changes of mustard gas injury of the eye

It may be that the corneal metabolism suffers by direct chemical damage and that this may happen in a clinically normal or nearly normal cornea. We know for instance that the cornea can recover more or less completely after a mustard gas injury. A breakdown of corneal tissue may occur many years after. I. Mann and B. D. Pullinger (*Proc. Roy. Soc. Med.*, Vol. XXXV, p. 229, 1942) have shown experimentally in rabbits that a lipoid change precedes the corneal breakdown. We do not want to go into details of the clinical or histological side of mustard gas poisoning but mention briefly the histological appearance of a late stage of corneal changes 27 years after the gas injury, as these changes fit well into our material and conclusions.

There was no clinical similarity between this case of greyish patchy opacities and a band-shaped corneal degeneration. We found in slides of the excised corneal opacity that Bowman's membrane (Fig. 11) was full of lime granules which were spread in a loose distribution in the anterior corneal lamellae. There was a whorled mass (Fig. 12) in front of Bowman's membrane which showed eosinophil staining and suggested the changes in band-shaped corneal degeneration described above. A calcified Bowman's membrane is fractured at many places. Calcification of Bow-

man's membrane in our case is a late sequel to mustard gas poisoning and may occur without corneal breakdown. We assume that the corneal damage by mustard gas poisoning may have been due to the pericorneal injury. Damage of pericorneal vessels is described in many fresh cases of mustard gas poisoning. There may have been a certain equilibrium achieved by corneal fluid exchange in spite of the reduced metabolism which may suffice to preserve corneal translucency. A slight slowing of the corneal metabolism creates the mechanism suggested above as the basic cause of calcification of Bowman's membrane and the secondary establishment of an eosinophil substance in front of the calcified Bowman's membrane. The nature of this process is necessarily progressive which is in accordance with clinical experience.

Conclusion

1. Precipitation of lime in Bowman's membrane in the form of calcium phosphate may be due to a slowing down of corneal metabolism. CO_2 escapes, alkalosis sets in and calcium phosphate is condensed in the tissue with the slowest metabolism which is Bowman's membrane. The influence of light in this process of precipitation is likely.

2. Calcified Bowman's membrane is an obstacle to the normal fluid exchange from epithelium to the anterior chamber. Its sequel is the establishment of an amorphous eosinophil substance in front of Bowman's membrane which was found in a case of band-shaped corneal degeneration and in a late, progressive case of macular corneal opacities after mustard gas poisoning.

3. Increased intra-ocular pressure may be a further obstacle to the entrance of fluid into the inner eye. The frequency of hemispherical bodies in Bowman's membrane in cases of primary and secondary glaucoma may be due to the precipitation at this border zone.

4. A case of arterial hypertension is described in which another form of purple staining concretions was present in Bowman's membrane and also in the superficial scleral layers. The precipitation may be a general metabolic deterioration of the ocular walls.

I am indebted to Professor A. J. Ballantyne of Glasgow and to Dr. Magnus of York for permission to use their material. Unfortunately black and white reproduction does not show the details present in the original coloured drawings. The work has been done in the Tennent Institute of Ophthalmology, University of Glasgow, and Professor W. J. B. Riddell has assisted in preparing the manuscript for publication.

AETIOLOGY OF IRIDOCYCLITIS IN THE AFRICAN*

BY

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WHILST stationed at Mwanza in Lake Province in 1944, I saw 42 cases of iridocyclitis in Africans. As soon as a diagnosis of iridocyclitis was made I attempted to persuade the patient to stay in hospital so that he could be under continuous observation and treatment. I often failed to gain acquiescence, and occasionally the patient took fright at the mere suggestion and I never saw him again. Nevertheless, I was able to investigate and treat 28 cases fairly thoroughly. The other 14 cases were not investigated fully and are not included.

This unwillingness to stay in hospital is readily understood. In the case of a man it means that his wife and family are alone and unprotected, a very real factor in Mwanza where thieving is so common. Moreover, " white " medicine is strange and the native is apt to regard disease as " shauri ya Mungu " (Fate or will of God) so that holding over his head the threat of losing his sight has little effect. Faith in the witch doctor dies hard. " White " medicine is tried only after native medicine has failed. I found one native just after he had been admitted to the ward chewing with great determination and discovered on close questioning that it was native medicine to protect him from harm whilst in hospital.

For the purpose of this article cases which would be diagnosed clinically as iritis or as cyclitis have been included under the one term iridocyclitis for convenience and also because, even when the inflammation appears to be confined to either the ciliary body or the iris, the other is bound to be affected.

Iridocyclitis may be :—

(1) Secondary to a local injury, inflammation or toxic process *e.g.*, a keratitis or scleritis or an intra-ocular pathological process such as a neoplasm or haemorrhage.

(2) Due to an exogenous infection such as a perforating injury or intra-ocular parasites.

(3) Due to an endogenous infection *e.g.*, syphilis, gonorrhoea, relapsing fever or a septic focus.

The aetiology of the first two groups gives rise to no difficulties but the last group of causes is far from being understood and it is with this last group that this article is concerned.

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All the observations on the cases were made with focal illumination, a corneal loupe and an ophthalmoscope. A slit-lamp was not available. The microscopic examinations were carried out as shown in Table II and no negative finding was accepted until several specimens had been examined on separate occasions but it was not possible to make more elaborate investigations such as cultures of the aqueous, urine, septic material from tooth sockets and swabbings from tonsils and sinuses, etc.

Comments on tables of cases

Although in each case I have recorded the period which the patient gave as the duration of the disease before he consulted me, I do not place great reliance upon these statements. The African lives from day to day with little thought for to-morrow or, perhaps it is truer to say, an indifference to the future. He is rarely really well and he is unlikely to notice a slight blurring of the vision of one eye and still more unlikely to come to hospital unless the pain is severe or the vision markedly diminished. Quite often the patient came complaining merely of pain and was most surprised to find during the examination that he was unable to see with the affected eye. I think it is probable, therefore, that the periods given in the table of the duration of the disease before treatment are considerably under-estimated.

The dearth of female cases of iridocyclitis in the above series is most noticeable. There was only one female patient (case No. 7) whom I was able to investigate at all fully, and there were two other female cases amongst the 14 patients who refused to stay in hospital. In one, an adult, the disease was almost certainly due to syphilis, and in the other, a young child, it was probably due to tick-fever since she said she had had a high fever a few weeks before and ticks were later discovered in her house. It is difficult to explain the preponderance of males. Undoubtedly the native woman's fear of leaving her husband and family plays a large part but this would explain the unwillingness to stay in hospital rather than the small number of women with iridocyclitis who were seen.

Notes on treatment

A brief account of the main lines of treatment may prove of interest. All treatment was made as routine as possible to avoid confusion because few of the African dressers were able to read very well and their capacity for forgetting to do any treatment out of the usual routine was extraordinary.

On admission the following treatment was given to every patient until the results of the investigations were received.

TABLE I—*Tabulation of Cases*

Name	Sex	Approx. age	Duration before treatment	Eye	V. before treatment	Tension of globe	Ciliary Inj.	Posterior Synechiae	K.P.	Period of treatment	V. after treatment
1. A.M. ...	Male	19	2 weeks	L.	F. at 2 M.	N.	Slight	None	Moderate	5 weeks	6/6
2. B.I. ...	Male	45	1 week	R.	F. at 2 M.	N.	Moderate	Marked	Moderate	3 weeks	6/24
3. B.Z. ...	Male	35	1 day	R.	6/60	+	Slight	Moderate	Slight	Refused admission	
4. C.H. ...	Male	28	2 days	R.	F. at 3 M.	N.	None	None	Marked	5 weeks	6/6
5. D.M. ...	Male	34	2 months	R.	6/6	N.	Slight	Moderate	None	Refused admission	
				L.	F. at 4 M.	N.	Slight	Marked	Moderate		
6. D.S. ...	Male	25	3 days	R.	6/24	N.	Slight	Slight	Moderate	3 weeks	6/9
7. G.M. ...	Female	12	2 weeks	L.	F. at 1 M.	N.	Slight	Slight	Moderate	Refused admission	
8. G.O.A.	Male	21	2 months	L.	6/9	N.	Slight	None	Marked	Ran away after 6 days	
9. H.A. ...	Male	23	1 day	R.	6/12	N.	Slight	None	Slight	Refused admission	
10. H.M. ...	Male	50	5 days	R.	3/60	N.	Moderate	Moderate	Marked	2 weeks	6/24
11. J.K. ...	Male	30	10 days	R.	F. at 3 M.	N.	Slight	Slight	Moderate	Ran away after 4 weeks	6/60
				L.	6/36	N.	Slight	Slight	Slight	,,	6/36
12. J.S. ...	Male	30	1 month	R.	6/60	+	Marked	Slight	Marked	3 weeks	6/9
				L.	6/36	+	Marked	None	Marked	3 weeks	6/9
13. K.T. ...	Male	50	4 weeks	R.	6/36	N.	Moderate	Marked	Marked	2 weeks	6/12
14. M.P. ...	Male	30	1 month	R.	F. at 3 M.	N.	Moderate	Moderate	Slight	3 weeks	6/18
15. M.K.	Male	25	4 days	L.	F. at 1 M.	N.	Moderate	Slight	Moderate	3 weeks	6/12
16. M.K.	Male	50	5 days	R.	H.M.	N.	Moderate	Marked	Moderate	3 weeks	6/24
17. M.K.	Male	14	1 day	R.	6/18	N.	Slight	None	Slight	4 weeks	6/6
18. M.M.	Male	30	4 days	L.	6/12	N.	Slight	Marked	Slight	Refused admission	
19. M.M.	Male	35	2 months	R.	F. at 4 M.	N.	Slight	None	Slight	4 weeks	6/6
				L.	F. at 2 M.	N.	Moderate	Marked	Moderate	,,	6/6
20. P.M. ...	Male	38	1 day	L.	6/12	N.	Slight	None	Slight	2 weeks	6/9
21. P.M. ...	Male	30	2 weeks	R.	H.M.	+	Moderate	Moderate	Marked	3 weeks	6/9

TABLE I—*Tabulation of Cases—(continued)*

Name	Sex	Approx. age	Duration before treatment	Eye	V. before treatment	Tension of globe	Ciliary Inj.	Posterior Synechiae	K.P.	Period of treatment	V. after treatment
22. P.K. ...	Male	20	2 weeks	R.	F. at 3 M.	N.	Moderate	Slight	Marked	2 weeks	6/12
23. R.B. ...	Male	40	2 weeks	R.	F. at 3 M.	N.	Moderate	Marked	Moderate	Refused admission	
24. S.K. ...	Male	50	5 days?	R.	F. at 3 M.	+	Moderate	Marked	Marked	5 weeks	6/24
				L.	F. at 3 M.	+	Moderate	Marked	Marked	,,	6/24
25. S.J. ...	Male	25	4 days	L.	6/60	N.	Slight	Moderate	Moderate	1 week	6/9
26. S.G. ...	Male	20	3 days	L.	F. at 1 M.	N.	Slight	None	Marked	3 weeks	6/18
27. S.M. ...	Male	50	5 days	R.	F. at 2 M.	N.	Moderate	Moderate	Moderate	3 weeks	6/9
28. S.B. ...	Male	30	1 month	R.	F. at 2 M.	N.	Slight	Slight	Slight	3 weeks	6/9

V.=Vision on Snellen's Test Type. F. at 1 M., 2 M.=Counted fingers at one Metre, two Metres, etc.
H.M.=Hand movements only. K.P.=Keratic precipitates. N.=Normal tension. +=Increased tension.

TABLE II—*Investigations*

Name	Kahn	Urethral Smear	Urine	Stool	B.S.	H.B.	Teeth	Tick fever	Remarks
1. A.M. ...	+	—	—	—	MP +	N.D.	Good	3 years ago	
2. B.I. ...	+ + +	PC + + + GC + +	—	—	—	75 per cent.	Good	None	
3. B.Z. ...	+	PC + + GC +	PC +	Strongyloides +	MP +	85 per cent.	Pyo +	None	
4. C.H. ...	+ + +	—	—	Taenia +	—	80 per cent.	Good	None	Had primary chancre 2 months ago.
5. D.M. ...	+ +	—	PC + +	—	—	90 per cent.	Good	4 months ago	Generalized enlargement of lymph glands.
6. D.S. ...	+ +	PC + + + GC +	SH + +	—	—	75 per cent.	Good	1 month ago	
7. G.M. ...	—	N.D.	—	—	—	75 per cent.	Good	None	

TABLE II—*Investigations—(continued)*

Name	Kahn	Urethral Smear	Urine	Stool	B.S.	H.B.	Teeth	Tick fever	Remarks
8. G.O.A.	—	—	RBC + + PC + +	—	—	70 per cent.	Good	None	
9. H.A. ...	+ + +	—	—	—	—	80 per cent.	Pyo + +	3 weeks ago	
10. H.M. ...	—	PC + + GC +	PC + +	Strongy-loides +	—	80 per cent.	Pyo +	None	
11. J.K. ...	+ +	—	PC + + RBC +	Ankyl +	—	45 per cent.	Pyo +	None	Spleen below umbilicus, developed mercury stomatitis.
12. J.S. ...	+ +	PC + + GC +	—	Ankyl +	N.D.	60 per cent.	Good	None	Generalized enlargement of lymph glands. Syphilis one year ago. Treated with 3 injections.
13. K.T. ...	+ + +	—	—	SM +	—	70 per cent.	Good	None	
14. M.P. ...	+ + +	PC + + + GC + +	—	Ankyl +	—	70 per cent.	Pyo +	None	Generalized enlargement of lymph glands. Fauces inflamed, phimosis and discharge.
15. M.K. ...	±	—	RBC + +	Ankyl +	MP +	80 per cent.	Good	None	
16. M.K. ...	+ +	PC + + +	PC + +	—	—	80 per cent.	Pyo +	None	Generalized enlargement of lymph glands.
17. M.K. ...	—	—	—	SM + Ankyl +	Sp + + +	50 per cent.	Good	18 days ago	
18. M.M. ...	+	—	—	Ankyl + Trichuris +	MPs +	90 per cent.	Good	None	
19. M.M. ...	+ + +	PC + + GC +	SH +	SM + Taenia +	MPs +	45 per cent.	Pyo +	None	Primary chancre and secondary rash present.
20. P.M. ...	+ +	—	RBC + +	SM +	—	90 per cent.	Pyo +	None	

TABLE II—*Investigations—(continued)*

Name	Kahn	Urethral Smear	Urine	Stool	B.S.	H.B.	Teeth	Tick fever	Remarks
21. P.M. ...	+ +	—	—	Ankyl + +	MPs +	80 per cent.	Good	None	Said he had syphilis 3 years ago. Penile scar present. Had relapse of iridocyclitis three weeks later which readily responded to treatment.
22. P.K. ...	+ + +	—	—	SM +	MPs +	N.D.	Good	None	Said he had syphilis 10 months ago.
23. R.B. ...	+ +	—	—	—	—	75 per cent.	Good	None	
24. S.K. ...	—	PC + + + GC +	—	SM + Ankyl +	MPs + +	75 per cent.	Pyo +	None	
25. S.J. ...	+ +	—	—	—	MPs + +	70 per cent.	Pyo +	None	Primary chancre, almost healed. Generalized glandular enlargement. Mucous papules in mouth.
26. S.G. ...	—	—	SH + +	—	N.D.	N.D.	Good	None	
27. S.M. ...	—	—	—	—	—	N.D.	Good	None	He had congenital cleft palate.
28. S.B. ...	+ +	—	—	—	—	90 per cent.	Good	None	

\+ =Present.
\+ + =Present in moderate numbers.
\+ + + =Present in large numbers.

N.D. =Not done.
G.C. =Gram negative intracellular diplococci.
PC =Pus cells.
RBC =Red blood cells.
SH =Schistosoma haematobium ova.
Ankyl =Ankylostoma ova.
SM =Schistosoma Mansoni ova.
MP =Subtertian malaria.
SP =Spirochaeta duttoni.

(1) An eyeshade made from blue paper and tape was worn constantly. Dark glasses were not available.

(2) The eyes were bathed thrice daily with hot water, using cotton wool bound on a wooden spoon.

(3) Atropine ointment (1 per cent.) was instilled at two hourly intervals during the first day and then, if the pupil was widely dilated, thrice daily. If the posterior synechiae were resistant the patient was given a subconjunctival injection of "mydricain" (a mixture of adrenalin, atropine and cocaine).

(4) The following blunderbuss mixture which has a foul and most persistent taste was given thrice daily. In the African's eyes the more "Kali" the medicine the greater its power and this mixture was greatly appreciated.

Re Potassium iodide gr. VII.
Sodium salicylate gr. XX.
Sodium sulphate 3I.
Solution of mercuric chloride M.XXX.
Water to 3I t.d.s.

(5) T.A.B. was injected intravenously on alternate days in doses of 0·5 c.c., 0·75 c.c., 1·0 c.c., and in some cases higher doses were given depending upon the patient's reaction and the condition of the eye.

(6) If the ocular tension was raised or the keratic precipitates were increasing a paracentesis of the aqueous was performed and repeated as often as it appeared necessary.

When the results of the various investigations were known any other diseases found were treated as indicated below. The treatments were occasionally modified when multiple infections were present.

Syphilis.—When the Kahn test was reported to be positive neoarsphenamine 0·3 gramme was given intravenously twice weekly in addition to the mercury and iodides by mouth.

Gonorrhoea.—In addition to the intravenous T.A.B. two tablets of sulphapyridine were given every four hours by day *i.e.*, ten tablets daily for five days.

Schistosomiasis (*Mansoni or Haematobium*).—Antimony tartrate grain I was given intravenously thrice weekly.

Ankylostomiasis.—For this disease 3 cc. of tetrachlorethylene combined with 1 c.c. of oil of chenopodium was given on an empty stomach in the morning followed by a brisk saline purge two hours later.

The multiplicity of possible endogenous infections

During the middle of the last century it was held that syphilis, tuberculosis and rheumatism were the chief causes of iridocyclitis. In more recent years, however, other diseases such as gonorrhoea, leprosy, bacillary dysentery and relapsing fever have been shown to cause the disease and the rheumatic group is now regarded on the Continent as allergic manifestations of tuberculosis, and by British and American Ophthalmologists as consequences of focal infections.

Duke-Elder, after summing up the evidence concerning this group, states that these cases, in his opinion, "are due to the excitation of a local allergic reaction by bacterial products liberated from some focus of infection in an immune but allergic patient." If this concept is true, then it follows that in any community where infections abound we should expect to find a high incidence of iridocyclitis. In my brief series of cases the multiplicity of infections from which the African in the Mwanza district suffers is in striking evidence and it appears that the incidence of iridocyclitis in this district is correspondingly high.

Comparison of series at three different stations

My figures for comparable series of African eye cases in Tanga, Mwanza and Iringa show an incidence highest in Mwanza and lowest in Iringa.

Place	No. of eye cases	No. of cases of iridocyclitis
Tanga	102	9 or 9 per cent.
Mwanza	252	42 or 16·1 per cent.
Iringa	323	6 or 1·85 per cent.

At both Mwanza and Tanga, malaria, syphilis, gonorrhoea, filariasis ankylostomiasis, schistosomiasis and relapsing fever are common. In Iringa, however, these diseases are much less frequently seen although trachoma is relatively more prevalent. Furthermore, in both Mwanza and Tanga, despite the greatest care in investigating and treating coincident infections, cataract extraction was a disheartening operation because so many eyes developed post-operative iridocyclitis and other complications and were lost. In Iringa, on the other hand, despite the fact that trachoma has usually been present I have had but one severe case of post-operative iridocyclitis in 34 cases to date. This is no doubt partly due to the cooler climate, but I am inclined to think that the chief cause is the relative absence of parasitic infections.

The Kahn reaction

Of my series of 28 cases, 20 showed a positive Kahn reaction, but the meaning of this seropositivity in the African is debatable. It is not proposed in this paper to enter into a discussion of the meaning of a positive Kahn reaction in the African, but the spirochaetal diseases are naturally considered largely if not entirely responsible. In Mwanza district, relapsing fever is common and appears to be on the increase, Weil's disease probably occurs, but florid yaws is now rarely seen in the locality. Primary and secondary syphilis are very common and I think that it can be assumed that most of the positive reactions in this series were due to syphilis.

Syphilis

Duke-Elder classifies syphilitic iridocyclitis into four groups:—

1. The commonest, occurring early in the secondary stage.
2. Recurrent, occurring as a relapse following inadequate treatment.
3. A form of Jarisch-Herxheimer reaction.
4. Late or tertiary manifestation.

Moore and Gieske have published a table of 249 cases of early, relapsing and late cases of syphilitic iritis which passed through their hands. This table shows that 95·5 per cent. of the early cases were associated with other manifestations of secondary syphilis. In the relapsing cases, 11·2 per cent. were accompanied by mucocutaneous relapse and 14·8 per cent. showed some form of neurosyphilis, but in 74 per cent. no associated lesions of any kind were discovered. Another table shows that the optic nerve was involved in 15·3 per cent. of the early cases, 24·1 per cent. of the relapsing and 8·2 per cent. of the late.

Parsons states that iridocyclitis occurring in the course of syphilis is an indication of a severe infection and quotes Trousseau who found that thirty-four of a group of forty such patients ultimately developed grave sequelae such as tabes dorsalis and general paralysis of the insane. It is interesting to note in this connection that tertiary syphilis in general and neurosyphilis in particular are not very common among the Africans despite the prevalence of syphilis.

In his book "The Modern Treatment of Syphilis" Moore stresses that the treatment of early and relapsing iritis must be designed to cure the syphilis completely, and it should be continued for a full year after the serological tests are negative and the eye has recovered. It is extremely difficult to reach this

standard with our African patients because they fail to understand the necessity for such prolonged treatment after the symptoms have gone, but we should keep this standard in mind. It seems inevitable at the present time that the great majority of Africans pass into a latent phase which might at any time relapse.

In my series only three cases presented with unequivocal signs of secondary syphilis (cases 4, 19, 25) but in five other cases there was a generalised enlargement of lymphatic glands or other evidence suggestive of syphilis. This leaves twelve cases with serological evidence as the only definite indication of syphilis although one of these (case 22) stated that he had had syphilis ten months previously. These figures suggest that many of my cases were in the latent phase.

None of my cases appeared to be a manifestation of the Jarisch-Herxheimer reaction and in this connection Moore considers that a reduction in the early dosage of neoarsphenamine is necessary only in those cases in which the optic nerve is affected.

Gonorrhoea

In nine cases there were numerous pus cells found in urethral smears and in eight of these Gram-negative intra-cellular diplococci were seen. These smears were obtained by a thorough massage of the bulb and shaft of the penis in the early morning. Prostatic massage was not done except in a few cases because I found that the natives objected strongly to this procedure and it usually frightened them away. I preferred to be less scientific and to keep my patients. Thus there is no doubt that many others had a gonococcal prostatitis or vesiculitis which was unfortunately missed. No cases were seen in the acute stage of gonorrhoea.

It seems to me that in these few cases gonococci were found in a relatively high number of patients. It is true that great care was exercised in searching for organisms under the microscope, but this is not an adequate explanation. I am inclined to think that the extreme drunkenness, sexual excess, and promiscuity which is so common in African towns like Mwanza, results in frequent reinfections and leads to a chronic gonococcal discharge which is not met with in British practice. In fact some African communities regard a moderate urethral discharge as an indication of manhood.

Two types of gonorrhoeal iridocyclitis may occur. One type appears characteristically about six weeks after the onset of the urethral discharge often in association with arthritis. It is usually severe and is believed to follow a gonococcal septicaemia. The second type is a late complication. It is less severe but tends to recur and is probably an allergic manifestation. None of these

cases was associated with arthritis, and I am inclined to think that the majority of them were of this latter group.

The treatment of gonorrhoea in an African is disappointing because no African will attend outpatients regularly when his pain or symptoms have disappeared. Prostatic massage and bougies are forms of treatment of which he strongly disapproves. In addition to this, there is the ever-present certainty of re-infection, so that I do not feel at all certain that any of these gonorrhoeal cases were really cured.

Tick fever or relapsing fever

Iridocyclitis is not a frequent complication of tick fever but when it occurs, it commonly does so towards the end of the disease or within three or four weeks of the last relapse when the blood is usually free of parasites.

If inquiry elicits a history of fever a native is ever ready in reply to a leading question to volunteer the information that his hut is tick infested and to agree that the fever was tick fever, but there is no means of confirming this. Indeed, in five cases only was it possible to prove from hospital records that the patient really had suffered from tick fever previously. In one of these cases it had occurred three years before and was therefore of no significance, and in another case it was four months earlier and so was of doubtful significance. Nevertheless, the disease is so common in Mwanza district that it may have been the cause of a few other cases in this series.

None of these patients developed optic atrophy which occasionally follows iridocyclitis in relapsing fever.

Dental sepsis

It is established that septic foci, such as infected teeth and nasal sinuses, give rise to iridocyclitis, the streptococcus being the commonest organism. Ten of these series had pyorrhoea alveolaris but unfortunately it was not possible to culture the organism or to give the patients any definite treatment.

In Africa no teeth lost can ever be replaced by false ones and consequently a native can be persuaded to part with teeth only if they are very painful. Thus apart from the removal of an occasional tooth and frequent phenol mouth washes, little was done for these cases.

Malaria

The blood slides of nine of these cases showed subtertian malaria parasites, but it must be borne in mind that every native in the

Mwanza district has a chronic malaria from constant re-infections which maintains his immunity at a high level. It is therefore not surprising that parasites were found in the blood of a few cases. Elliot quotes Smith, who had a few cases of low-grade iritis, and Duke-Elder quotes several other authors who have recorded cases of serous iridocyclitis which they thought to be of malarial origin. Both authors, however, are very sceptical of the importance of malaria in causing iridocyclitis. Elliot points out that malaria is such a common disease in tropical and sub-tropical countries that it is surely of significance that in the voluminous literature upon malaria there is such scant reference to ocular complications and sequelae. It is, therefore, doubtful whether malaria was a factor in causing the iridocyclitis of these cases except possibly by reducing the general resistance of the body.

Other possible aetiological factors

In three cases schistosoma haematobium eggs were found in the urine, in six cases schistosoma Mansoni ova were seen in the stool, eight patients had ankylostomiasis and two patients had taeniasis. How often, if ever, do these diseases give rise to iridocyclitis?

Ophthalmologists are agreed that septic absorption from the alimentary canal may sometimes cause anterior uveitis, and in this connection streptococcal oral sepsis, gastro-enteritis especially in children, non-dysenteric amoebiasis, cholecystitis and appendicitis have been cited as aetiological agents. Iridocyclitis is a well known complication of bacillary dysentery and it usually occurs about three weeks after the onset of the disease often in association with arthritis. It has also been recorded as a complication of amoebic dysentery. Schistosoma Mansoni infections often present with symptoms resembling those of bacillary dysentery and it is possible that bilharzial dysentery may occasionally give rise to iridocyclitis. The pathology of ankylostomiasis has not been fully worked out, but many clinicians believe that the anaemia and debility of this disease are due not only to loss of blood but to a continuous absorption of both specific toxins elaborated by the worms, and to toxins passing from the alimentary canal through the damaged intestinal mucosa. Thus ankylostomiasis may act as a factor in producing disease of the anterior part of the uveal tract.

Infections of the genito-urinary tract play some part in the aetiology of uveitis and, although gonorrhoea is the most common cause, streptococcal infections of the prostate and posterior urethra have been proved to be aetiological agents in some cases. Bilharzia may attack any part of the genito-urinary tract and thus give rise

to cystitis, prostatitis and pyelitis, so one is tempted to wonder whether this disease may be a factor in causing iridocyclitis in Africans.

In any case all the above mentioned diseases give rise to the absorption of toxins and, even if they do not cause iridocyclitis directly by sensitizing the uveal coat so that a later escape of toxins gives rise to allergic inflammation, there is the possibility that they may act indirectly by reducing the body's general resistance. It must be conceded, however, that this series gives no grounds for the belief that these parasitic diseases are common causes of uveal affections, for in two cases only, *viz.*, case Nos. 4 and 26 did any of these diseases occur alone.

It should be mentioned that leprosy, trypanosomiasis and onchocerciasis may cause inflammation of the uveal coat although none of these diseases was seen in my series of cases.

The incidence of typhoid fever in a community is considered to be a good index of its standard of sanitation, and it may be that the incidence of iridocyclitis in a tropical community is an index of the extent to which it is disease-ridden. Much work will need to be done before the part played by tropical diseases in the aetiology of iridocyclitis can be properly assessed, but it is hoped that this paper will draw some attention to this subject even though it does little to elucidate the problem.

Summary

Twenty-eight African cases of iridocyclitis seen in Mwanza, Lake Province, were investigated with the following results :—

Kahn reaction	20 cases positive
Urethral smear	9 showed pus cells of which 8 showed gonococci
Tick relapsing fever	3 definitely associated with iridocyclitis
Dental sepsis	Present in 10 cases
Malaria	Subtertian parasites present in 9 cases
Schistosoma Haematobium ...	Eggs present in urine of 3 cases
Schistosoma Mansoni	Eggs present in stools of 6 cases
Ankylostomiasis	Present in 8 cases
Taeniasis...	Present in 2 cases

The importance of the above diseases in the causation of iridocyclitis in the African is discussed.

In conclusion I wish to record my especial thanks to the Senior Specialist, Dr. W. A. Young, for the encouragement he has given me in this work, for much constructive criticism and in particular for his help in supplying the references quoted in the section dealing with syphilis.

I am indebted to the Director of Medical Services, Tanganyika Territory, for permission to submit this paper for publication.

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AMNIOTIC MEMBRANE GRAFTS IN CAUSTIC BURNS OF THE EYE*

(Burns of the second degree)

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THAT caustic burns of the eyes produce more ultimate damage than the immediate reaction would suggest and that recovery is always protracted is a common experience. To overcome corneal damage developing during the course of the affection, and to avoid conjunctival complications as exemplified by symblepharon, a variety of procedures have been suggested. Neutralization of the caustic appears to be of little use, whilst for severe burns immediate excision of the damaged conjunctiva, as advocated by Denig, seems to be valuable. The pathology of caustic burns, is, however, far from clear. How much of the damage is produced instantaneously and irreversibly is not known, nor is it known how

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lesions in the cornea come to be developed as late manifestations. These might conceivably be delayed reactions to the instantaneously acting irritant, or the sequel to irritant products of the conjunctival lesion. Though there are many experimental studies on these points, no clear conclusions appear to be possible.

This lack of precision is reflected in the therapeutic measures. The Denig procedure is apparently based on the assumption that irritants liberated by the necrosed conjunctiva are the essential noxious agents. A. L. Brown (*Arch. Ophthal.*, Vol. XXVI, p. 754, 1941) holds, on clinical and experimental evidence, that the apposition of inflamed surfaces tends to maintain the inflammatory reaction and advocates separating the opposing surfaces by a graft of fat-free peritoneum from the rabbit. Both Bulson and Masters in discussing Brown's paper from personal experience supported his procedure but stressed that rabbit's peritoneum tended to act as an irritant.

Clinical findings in 30 cases of caustic burn treated by grafting with amniotic membrane

Brown's suggestion that inflamed surfaces should be kept apart seems clinically sound. It was felt that amniotic membrane of human origin might be more suitable than rabbit's peritoneum. An opportunity to test this presented itself in a patient who responded but poorly to the classical methods of conservative treatment.

Case report. T.D., aged 50 years, ten days ago sustained a moderately severe lime burn which had involved the upper and lower fornices and the lower third of the cornea. He had been receiving the usual in-patient treatment including atropine, irrigations, and the daily passage of a glass rod round the fornices to prevent symblepharon formation. On the tenth day the lower third of the cornea still stained with fluorescein and the bulbar and palpebral conjunctiva of the lower fornix showed the ulcerated and marbled appearance typical of this type of burn. An amniotic membrane graft was applied so as to separate the affected conjunctival surfaces. The eyes were bandaged for two days and at the end of that time it was found that the whole appearance of the burn had altered considerably. The conjunctiva had become more normal in colour, had a smooth surface and no longer stained with fluorescein. The corneal abrasion had also healed. Observation was continued for a few days; vision was now 6/6 and as the eye was white no further treatment was found to be necessary.

This encouraging result led to a more extensive trial of the method. To date thirty burns have been treated. The details are shown in Table I and the following summary gives the essential features in this series:—

TABLE I

Clinical details on caustic burns healed by amniotic graft

Successive No. in series	Age	Extent of cornea involved	Conjunctival fornices involved	No. of days when grafted after burn	Days in hospital	Final vision (with correction)	Nature of caustic	Remarks
1	50	Lower 1/3	Upper and lower	10	17	6/6	Lime	—
2	20	Lower ½	Upper and lower	4	9	6/5	Lime	—
3	40	Complete	Upper and lower	6	21	6/9	Lime	Lime 4 days in fornices
4	64	Lower 1/3	Lower	2	10	6/9	Lime	—
5	52	Lower 1/3	Lower	2	8	6/9	Lime	—
6	30	None	Lower	2	5	6/6	Hydrochloric acid	—
7	16	Lower 1/3	Lower	1	5	6/5	Lime	—
8	17	Lower 1/3	Lower	2	7	Not recorded	Lime	—
9	25	Lower ½	Upper and lower	2	12	6/5	Lime	—
10	56	Lower ½	Upper and lower	3	17	Not recorded	Lime	—
11	43	Lower ½	Upper and lower	5	18	6/9	Lime	—
12	64	Complete	Upper and lower	5	64	P.L.	Ammonia	—
13		Complete	Lower	6	64	6/6	Ammonia	—
14	16	Lower ⅔	Upper and lower	3	10	6/6	Sodium hydroxide	—
15	39	None	Lower	2	5	6/6	Hydrochloric acid	—
16	15	Lower ½	Lower	3	15	6/9	Lime	One strand of symblepharon
17	50	Lower ½	Lower	2	10	6/6	Lime	—
18	50	Lower ½	Lower	2	6	6/6	Lime	—
19	37	Lower ½	Lower	3	11	6/6	Lime	—
20	17	Lower ½	Lower	5	7	6/6	Sodium hydroxide	—
21	48	Complete	Upper and lower	3	27	6/6	Lime	Two grafts (on 3rd & 35th day)
22	24	Lower ⅔	Upper and lower	2	2	6/6	Lime	—
23	36	Lower ½	Lower	0	—	6/6	Lime	Grafted in out-patient dept.
24	32	Lower ½	Lower	0	5	6/6	Lime	ditto
25	47	Lower ½	Lower	0	7	6/12	Lead	ditto
26	14	½	Lower	0	2	6/6	Lime	—
27	39	Lower ½	Upper and lower	0	4	6/6	Lime	—
28	38	Lower ⅔	Lower	3	9	6/6	Sulphur dioxide liquid	—
29	16	Lower ½	Lower	2	5	6/6	Lime	—
30	38	Lower 1/3	Lower	0	—	6/6	Lime	Graft done in out-patient dept.

1. Nature of caustic:—Hydrochloric acid and sodium hydroxide were each responsible for 2 cases; liquid sulphur dioxide, and lead for one each, whilst ammonia caused burns in the two eyes of the same patient. In the remaining twenty-two cases lime was the responsible agent.

2. Severity. In all cases except two—the exceptions being the two burns caused by hydrochloric acid—the cornea was involved when the patient was first seen. Mild cases without corneal involvement were not subjected to grafting. Corneal involvement at its mildest was loss of epithelium as shown by staining with fluorescein; severe involvement, as shown by diffuse or localised opacification of the cornea, was less common. Localised reddening of the conjunctiva at the site of the burn was a constant feature, and fluorescein staining revealed the extent of the epithelial damage. The severity of the burns in this series can be judged by the following data:—

Conjunctiva:	
Both upper and lower fornices burnt ...	11 cases
Lower fornix only	19 cases
Cornea:	
Clear	2 cases
The whole of the cornea involved	4 cases
Three-quarters of the cornea involved ...	3 cases
Half of the cornea involved	11 cases
Lesser areas	10 cases

3. Time of grafting. In the earlier cases a graft was generally not applied until after some days. More lately grafting has been carried out immediately.

4. Duration of treatment and end-result. At first the patients were admitted to hospital. More lately immediate grafting has been carried out in the out-patient department with or without admission for 2 to 3 days as in-patients. To present averages in this experimental series would be fallacious, but it may be stressed that immediately treated eyes and those treated early, tend to be very nearly white in 3 to 5 days; by the end of a week the eye is generally normal. In no case, except the three specially noted below, were there any corneal or conjunctival sequelae. In twenty-eight eyes in which vision is known, twenty-six showed vision of 6/9 - 6/5.

5. Complications and sequelae. In three cases in this series of thirty permanent complications were noted.

(1) One thin strand of symblepharon developed in case 16. It does not appear to give any difficulties or discomfort.

(2) Irregular cloudiness of the cornea has persisted in case

3 (in which the lime had stayed in the fornices for four days before treatment was sought).

(3) Cases 12 and 13. A man, aged 64 years, sustained a severe ammonia burn in both eyes, distinctly more marked in the right eye than the left. He did fairly well after two evacuations of the anterior chamber of the right eye, but on the tenth day developed hypopyon which did not respond to treatment. An amniotic graft had been inserted on the fifth day. The eye must be regarded as lost from inflammatory reaction. This patient, incidentally, illustrates the value of amniotic grafting in cutting short complications that had already set in. Whilst the right eye caused anxiety from the beginning, the left appeared at first to show an uncomplicated recovery. By the sixth day, however, fairly tense strands of symblepharon had formed, which reformed after breaking down with a glass rod, and it was then that an amniotic graft was applied. On removing the bandage forty-eight hours later it was gratifying to find a practically normal conjunctiva without any trace of symblepharon formation. This eye is now—six months later—completely normal. (In another patient treated since this series was closed, an amniotic graft applied on the fifth day in a cement injury, which had not been grafted initially, also controlled a tendency towards symblepharon formation.)

6. Delayed recovery. In one patient (No. 21) recovery was distinctly slow. The whole of the cornea was diffusely opacified and a graft was not applied until after he had been treated for three days by another surgeon. Both fornices were involved, and twenty-seven days of in-patient treatment was needed. Even then the eye was irritable. A second graft applied in the out-patient department on the thirty-fifth day gave a remarkable improvement within two days. The eye is now quiet and vision is full 6/6.

Technique of grafting

The human amniotic membrane ("amnioplastin") is that prepared by the London Hospital. It is dry and free from fat and resembles tissue paper. It is sent out in pieces of about four square inches folded twice and kept sterile in a cellophane envelope inside the usual paper covering. The folded piece is roughly 1" by 1" and this is about the right size when both fornices are involved; if only one fornix is to be grafted the membrane may be folded again. Several layers are an advantage as the tissue is friable and the needle tends to cut out if too few layers are used. It was found that the material is more easily handled dry as it tends to crumple and become unmanageable when it becomes wet. For this reason the tissue is folded to the correct size and then placed on a sterile dry towel and a No. 6 ophthalmic needle threaded with

No. 0 black silk is passed through each corner. The towel with graft and threaded needles is then brought up to the eye which is already cocainised and kept open by a self retaining speculum. The needles are then in turn passed through the bulbar conjunctiva and the graft sutured into place. When the fornix alone is involved it is sufficient to place two sutures in the depths of the fornix and the other two near the limbus so that all the affected bulbar conjunctiva is covered as well as the lower section of the cornea.

It is not advisable to cover too much of the corneal surface as the presence of the graft appears to affect the cornea adversely and

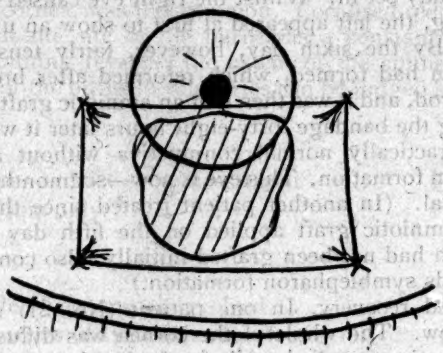


FIG. 1.

Schematic representation of graft held in position by four sutures (two in the lower fornix and two on the bulbar conjunctiva) and covering a burn involving the lower third of the cornea and a larger area of conjunctiva. (Area of burn shown by shaded lines.)

there is a temporary increase in the haziness of the cornea covered. This was noted in early cases when the whole cornea was covered and did not appear later when this point was appreciated. When it is necessary to put in a graft from the depths of the upper fornix to the depths of the lower fornix which necessarily passes right over the cornea, it is advisable to cut a small circular opening in the graft to correspond to the central area of the cornea.

It was at first thought that the optimum time for insertion of the graft was on about the second or third day after the burn, but it appears that immediate grafting is best. When the bandages are removed after 48 hours it is found that the graft is no longer visible. It has become transparent and forms a layer over the conjunctival surface. It there are any tags which have not been well in contact with the conjunctiva these may be seen as semi-transparent pieces of membrane in the conjunctival sac. The previously ulcerated conjunctiva is invariably found to have a smooth surface

and does not stain with fluorescein and though there may be some localised temporary corneal haze there is most often no staining of the corneal surface. The sutures are removed on the third or fourth day and at this stage the eye can be left open and protected with parolein drops and dark glasses, or if there is still some irritation a light pad can be used for a few days. The patients were not discharged until the eyes were quiet and there was no staining of cornea or conjunctiva.

To get the best results it is probably necessary to bandage both eyes for 48 hours as this ensures close apposition of the graft to the conjunctiva. In moderately severe cases a uniocular bandage has been found adequate.

Prior to inserting the graft the eye should be subjected to intensive local penicillin therapy. Penicillin drops in a concentration of 2,500 units per c.c. should be used. Where the graft is done immediately and in the out-patient department it is best to instil one drop every minute for ten minutes. For delayed grafting in the in-patient department more leisurely disinfection of the conjunctiva can be carried out.

Experimental study

Whilst these clinical investigations were in progress an attempt was made to study the effect of amniotic grafting under experimental conditions in the rabbit. These studies were largely unsuccessful owing to a combination of two circumstances. In the first place it was difficult to obtain a standard control lesion by the use of lime, of which several varieties, such as slaked lime and chalk lime, were tried. Small quantities applied to the conjunctiva tended to produce lesions that healed spontaneously, whilst massive applications destroyed the eye. No adequate dosage for a controlled lesion giving scarring of the cornea could be established. Something approaching a controlled standard lesion was obtained when the upper fornix and the upper bulbar and tarsal conjunctiva were painted with 20 per cent. silver nitrate, care being taken to wash away any excess before the lids came in contact with the cornea. Even so, a contact burn tended to form on the cornea, but in addition there appeared to be a corneal lesion secondary to the conjunctival damage. An even more serious difficulty emerged when amniotic grafting was attempted on to these relatively satisfactory lesions. It was found that the membrane behaved as an irritating foreign body. Instead of becoming readily absorbed, as it does in man, it tended to swell and assume a hard parchment-like aspect. The longer it was left in the eye the more irritation it caused, and if removed early it did not seem to have much effect one way or the other. It is possible that amniotic membrane of rabbit origin would behave differently, but this point was not pursued.

Discussion

In the absence of control clinical or experimental evidence, the results recorded here must be taken as an expression of clinical opinion rather than proof. The protracted course of caustic burns and the tendency towards conjunctival and corneal complications are, however, well known and the rapid recovery after grafting, both in fresh cases and in cases that have shown poor response to the standard treatment, is impressive. Simple as grafting with amniotic membrane is, and satisfactory as the results are, it is not suggested that all caustic burns are best treated by grafting with amniotic membrane. Patients whose cornea shows minimal or no fluorescein staining some hours after experiencing a caustic burn—burns that might be designated as those of the first degree—are quite safely treated by the classical methods, and they respond quickly. Patients with severe lesions with necrosis and blackening of the conjunctiva (burns of the third degree) appear to be best treated on the lines laid down by Denig, but of this type of burn and the appropriate therapeutic procedure we have no experience. Most caustic burns are intermediate between these two types in severity, and may be designated as burns of the second degree. These show a localised and relatively superficial conjunctival lesion and a variable superficial corneal lesion. In these cases, which are both common and productive of much disability, grafting with amniotic membrane appears to offer a simple means of considerably reducing the normally protracted course of recovery and preventing both corneal and conjunctival complications.

No opinion is offered on the mode of action of amniotic membrane grafts. It is possible that the graft acts in the manner suggested by Brown for the use of grafts of rabbit peritoneum, *i.e.*, mechanical separation of burnt surfaces; but it is difficult to believe that any substantial mechanical effect can be achieved by amniotic membrane which is rapidly absorbed. That amniotic membrane grafts act biologically rather than mechanically is suggested by the rapid effect and still more by the fact that it behaves in an apparently species-specific manner, if one is to judge by the different behaviour of the graft in man and in the rabbit. In one patient the graft became loose and was found 24 hours later under the bandage on the skin of the lid. On the conjunctiva it would have become largely absorbed by that time; on the skin it was similar to the appearance an amniotic graft assumes in the rabbit's conjunctival sac. This, too, rather suggests a specific biological interaction between the graft and the damaged conjunctiva.

Summary

1. An account is given of thirty cases of caustic burn of the eye treated by grafting with human amniotic membrane. Recovery was rapid and there were but few complications and sequelae.

2. The technique of amniotic membrane grafting is described.

3. Grafting with amniotic membrane—immediately or after 1 to 2 days—is advocated for second degree burns of the eye.

4. The difficulties of assessing experimentally the value of amniotic grafting are indicated and the possible mode of action is discussed.

QUININE AMBLYOPIA*

BY

J. D. J. FREEMAN

GLOUCESTER

THE following case history demonstrated several interesting points and for that reason is worth recording. The patient, Mrs. L., was a leading Wren aged 27 years, and was stationed at a Royal Naval Establishment on the east coast of Ceylon. She was married in June, 1944, and went up country for her honeymoon. Towards the end of this period, July 2, she developed general symptoms of malaise, frontal headache, pain behind the eyes and on moving the eyes, shivering at times and teeth chattering. On her return to Trincomalee on July 4 she was admitted to the sick quarters where her temperature was found to be 104° F. As well as general malaise she was complaining of headache, backache and nausea. She had left England in November, 1943, and had had no previous tropical diseases. I did not see her until July 24, 1944, when she was admitted to R.N. Auxiliary Hospital, Colombo, and came under my care. The following physical signs up to that date are taken from the notes made by Surg.-Commander H. L. Hoffman. R.N.V.R., Medical Officer in charge at Trincomalee.

General examination revealed no abdominal tenderness or enlargement of liver or spleen. On July 5 her urine contained bile pigments, but no albumen. On July 8 a blood film showed the presence of B.T. malaria parasites. The same day she started quinine and was given grs. x t.d.s. by mouth. On July 11 her spleen was palpable and she was also tender in the gall bladder area. Two days later on July 13 she complained of deafness and of seeing black spots in front of her eyes, which became bigger and paler turning greenish in colour. Her quinine therapy was stopped at once. She had had 120 grs. in all. She was put on tabatebrin until July 18th being given 1·2 grammes in all. On July 19 she was complaining of blurred vision, especially in the left eye and that her eyes were sore and hurt on movement. The next day her pupils were dilated and reacted sluggishly to light. On

* Received for publication, March 8, 1946.

July 21 her pupils were widely dilated. The left pupil did not react directly to light and the right pupil only reacted poorly. The left vision was no perception of light, and the right vision was counting fingers across the ward. The optic discs were swollen and there was a small flame shaped haemorrhage below the right disc. The external ocular movements were normal. There was no deafness now, blood pressure was 120/60, but she was complaining that her feet felt cold. On the following day the coldness had spread up to her knees. Her right vision was now only counting fingers at one metre. The retinal arteries were constricted and the veins engorged. There were some vague parasthesiae in the legs and the following rather indefinite C.N.S. signs—blunting to pin prick in lower part of legs—poor sense of position in the right big toe—equivocal plantar responses and absent abdominal reflexes. She was put on tab. trinitrin grs. 1/100 six hourly; tab. digitalis leaf (grs. 1½) six hourly, given alternately at three hourly intervals.

On July 24 she was admitted to R.N. Auxiliary Hospital, Colombo, where she came under my care. Her pupils were three quarter dilated, circular, inactive to light, but reacted on convergence. There was no perception of light in either eye. The media were clear. The fundi looked pale and oedematous, veins engorged and arteries constricted. The edges of the discs were blurred, but there was no measurable swelling. The small haemorrhage just below the right disc already mentioned was still present.

The subsequent course of events was as follows:—

July 27. Her discs appeared rather less swollen, but she still had no P.L. The trinitrin was stopped as it seemed to be upsetting her.

July 29. Examined by the Medical Specialist (Surg.-Lieut.-Comdr. J. H. Cobb, R.N.V.R.), who found she had severe circulatory disurbance of hands and feet which were pallid and cold. The peripheral nerves showed no defect. The abdominal reflexes were reduced but present, and plantars were flexor. He found no evidence of other neurological disease and thought acute disseminated sclerosis could be ruled out. Lumbar puncture and blood count were carried out and showed no abnormality apart from some anaemia.

July 30. She thought she could see slightly with her right eye; her pupils and fundi appeared unchanged.

August 1. Right pupil reacted briskly to light—vision hand movements in central field—field very constricted especially down and out. No P.L. in left eye and left pupil inactive directly to light.

August 2. Blood urea estimated 34 mgms. per 100 c.c.

August 3. Left pupil reacting to light directly, but not brisk. Vision hand movements in both eyes. Discs seemed less swollen and temporal edge of left disc could be made out.

August 5. Discs definitely less swollen, temporal edges of both discs seen. Vision: right counting fingers at one metre, left counting fingers at one foot. Right pupil maintained its reaction to light well, but not the left. Discs clearer and arteries appeared less constricted. The retinal haemorrhage in the right eye had now absorbed. On confrontation, fields very constricted in lower temporal areas, practically to fixation in left eye.

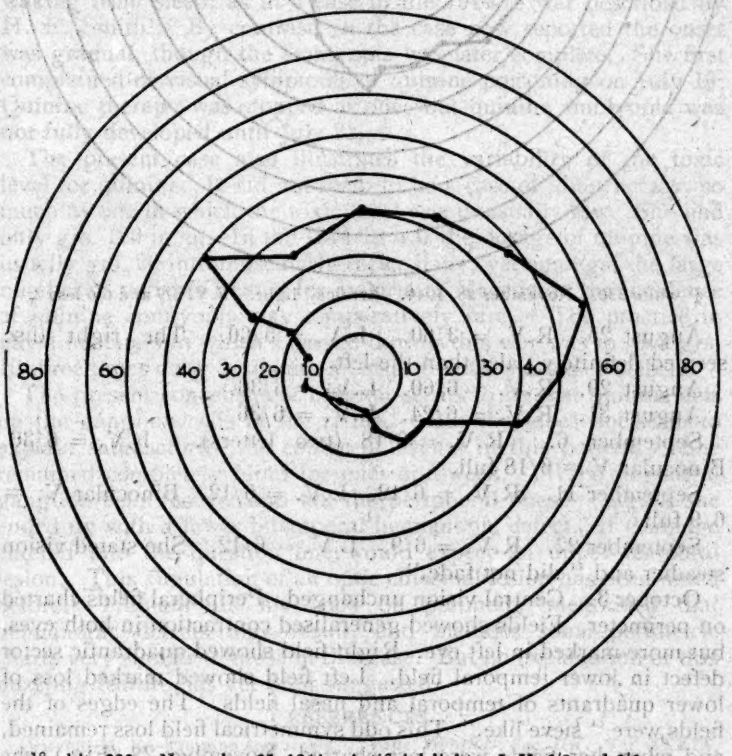
August 13. Could now see to get about a bit, although vision seemed unchanged. Hands better colour and not cold to touch, but feet still cold to touch.

August 15. R.V. = 2/60. L.V. = Counting fingers at one foot.

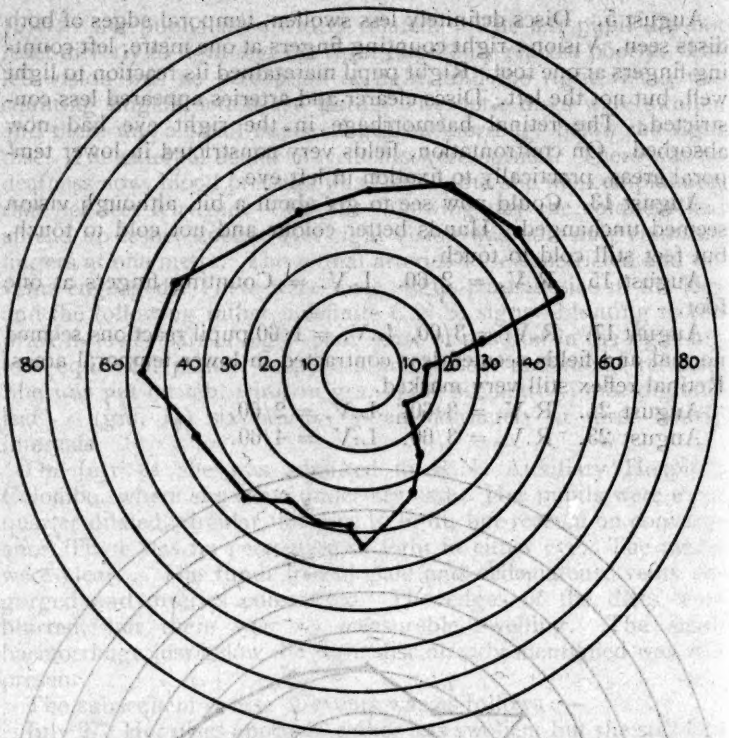
August 17. R.V. = 3/60. L.V. = 1/60 pupil reactions seemed normal and fields seemed less contracted in lower temporal areas. Retinal reflex still very marked.

August 21. R.V. = 3/60. L.V. = 2/60.

August 23. R.V. = 3/60. L.V. = 4/60.



½° white 330. November 25, 1944. Artificial light. L.V. 6/9 and 6/6 some.



½° white 330. November 28, 1944. Artificial light. R.V. 6/9 and 6/6 less 2.

August 25. R.V. = 3/60. L.V. = 5/60. The right disc seemed definitely paler than the left.

August 29. R.V. = 6/60. L.V. = 6/36.

August 31. R.V. = 6/24. L.V. = 6/36.

September 6. R.V. = 6/18 (two letters). L.V. = 6/36. Binocular V = 6/18 full.

September 11. R.V. = 6/12. L.V. = 6/12. Binocular V. = 6/9 full.

September 22. R.V. = 6/9. L.V. = 6/12. She stated vision steadier and "did not fade."

October 3. Central vision unchanged. Peripheral fields charted on perimeter. Fields showed generalised contraction in both eyes, but more marked in left eye. Right field showed quadrantic sector defect in lower temporal field. Left field showed marked loss of lower quadrants of temporal and nasal fields. The edges of the fields were "sieve like." This odd symmetrical field loss remained, and when her fields were last charted, November 28 (Fig.), she

still had this loss down and out but more marked in the left eye. A stereoscopic X-ray of skull showed no abnormality.

October 24. She developed dengue fever.

November 10. She had a recurrent malarial attack. B.T. shizonts were demonstrated in her blood and she was treated with tab. atebrin.

December 4. R.V. = 6/6 (less two). L.V. = 6/6 some letters. Binocular vision 6/6 full. Her discs were definitely pale but vessels seemed normal in calibre though the arteries might still be slightly constricted. Just prior to her return to England R.V. = 6/6 full, L.V. = 6/6 full and binocular vision 6/5 (less two).

Discussion

There are several very interesting points about this case. Most writers on quinine amblyopia have emphasised the suddenness of its onset. Often the first indication has been the patient's complaint that he has suddenly become blind—perhaps immediately after waking from sleep, as in a case in the 1914-18 war described by H. E. Smith[1]. By contrast, in the case now reported the onset was gradual, though the amblyopia was later complete. She first complained of visual symptoms of quinine poisoning on July 13. Quinine therapy was stopped at once but quinine amblyopia was not fully developed until July 21.

The present case also illustrates the variability of the toxic level for quinine. It did not seem to be a case of idiosyncrasy, so much as one in which the toxic level was unusually low. She had only grs. 120 in all. In the 1914-18 war the dosage of quinine was usually grs. 20 intramuscularly thrice daily, yet amongst the large number of patients treated for malaria in Macedonia the incidence of quinine amblyopia was comparatively rare. The practice in naval hospitals in which I served during this war was to give grs. 10 three times daily by mouth alternated with courses of atebrin.

The present concensus of opinion seems to be that quinine acts on the ganglion cells of the retina. But that does not seem to explain satisfactorily the course of events in this patient. She remained completely blind for over one week. Why do central ganglion cells recover and not the peripheral ones? Again she ended up with a lower bitemporal hemianopic defect. If drainage entered into the question one would expect an upper temporal lesion. This simulation of an optic chiasmal lesion has been noted before. Traquair[2] in his book on perimetry mentioned that hemianopic defects may result, and includes some perimetric charts very similar to those in this case. But no explanation of this puzzling feature has yet been suggested.

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AN EXOPHTHALMOMETER FOR DIRECT MEASUREMENT*

BY

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NEUROSURGEONS and physicians no less than ophthalmologists are conscious of the difficulties in eliciting minor degrees of exophthalmos or changes in an already known proptosis. The instruments in common use are not beyond criticism, for it is not unusual for different observers to obtain different readings in the same patient.

The ingenious apparatus devised by Hertel is widely used in spite of its being rather bulky, but it is generally agreed that it does not provide a very accurate reading as it relies on indirect measurements, *viz.*, not the position of the globe itself, but its corneal image in a mirror alongside a millimetre scale. The position of the head of the observer, and other factors such as the shifting gaze of the observer and possibly also movements of the patient's eye influence the readings, so that inconstancies of 1 mm. or more

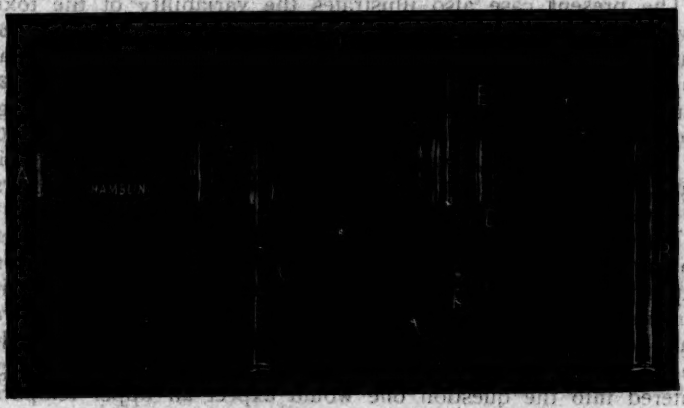

FIG. 1.

are not infrequent. To obviate these difficulties a variety of instruments have been made on the principle of the Luedde exophthalmometer. They all rely on a transparent rule which is placed on the temporal orbital rim; the corneal apex can be seen through the

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rule and its prominence measured. These small instruments are inexpensive, easy to carry and to use, but owing to the fact that the cornea and the rule cannot be brought close together, the results they give are not superior to those obtained with a Hertel exophthalmometer. The instrument designed by Mutch of Aberdeen has the merit of recording a direct measurement. It aims at measuring the projection of the eye beyond the level of a plane formed by the upper and lower orbital rims at their vertical centre over the closed eyelids. Possible sources of inaccuracy in its use arise from the fact that it is employed without reference to any clearly defined bony landmark, and that it has to be placed over a variable thickness of soft tissue, not necessarily identical on the two sides.

A simple means for direct exophthalmometry devoid of some of these pitfalls appears to be provided by the following design, which relies on measuring the forward position of the globe in relation to the fixed and easily identified outer bony canthi of the orbit.

The instrument (Fig. 1) consists of a measure rule (A) carrying a fixed vertical stem (B) and a movable stem (C) sliding along the rule. Between the stems B and C there is a slide (D) carrying a plunger (E) calibrated in millimetres, and ending in a cup (F) resembling that part of the tonometer which fits on to the cornea. Both stems (B and C) end in pads that can be made to rest on the external orbital margins, outside the outer canthi.

Mode of use

The preparation of the patient and the position of the observer are as with tonometry readings. The patient may be prone on a couch or sitting up with his head thrown back, whilst the surgeon stands behind. The corneae are anaesthetised, also as for tonometry. The surgeon adjusts the distance between the stems B and C so that their pads rest on the two external orbital canthi. Firm pressure must be applied on the instrument in order to get a constant and equal maximal depression of the skin on the two bony canthi. Then, before making the patient open his eyes the surgeon slides the plunger (E) so that it approximately centres over one of the corneae (Fig. 2). The patient is now asked to open his eyes and to direct his gaze straight to the ceiling. The cup (F) on the plunger is then brought down on to the cornea so that it covers it centrally (Fig. 3). A direct reading is now taken on the scale on the plunger. With the instrument still in position the plunger (E) is raised from the cornea and slid along the horizontal bar (A) so as to bring it over the cornea of the other eye, the position of which is now measured.

In order to be certain not to vary any factor in subsequent

measurements it is convenient to record the intermarginal distance (B to C) and to apply it in further readings.

In no normal case has the summit of the cornea as determined by these readings been less than 12 mm. above the fixed bony landmark of the outer orbital canthus. Generally the reading is 14-16 mm. No opportunity has presented itself for using the apparatus

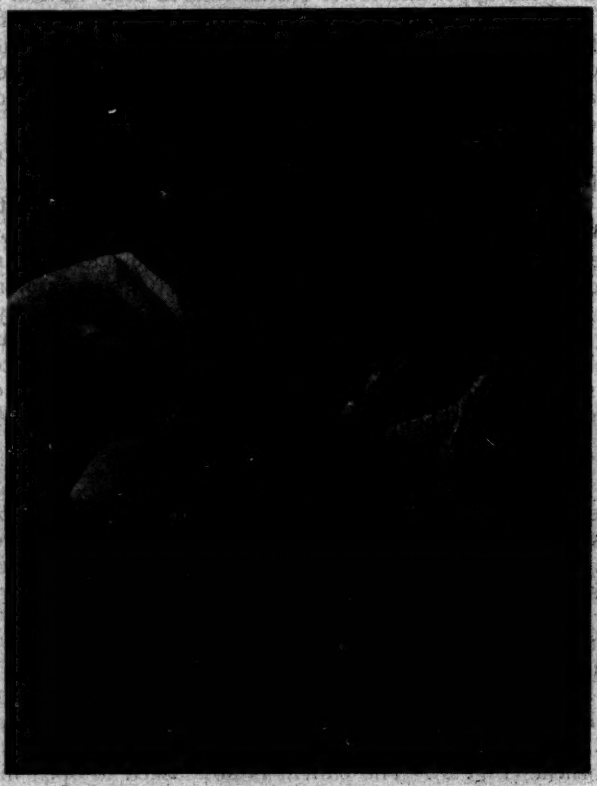

FIG. 2.

for the measurement of enophthalmos, but there are no theoretical reasons against such a procedure.

Sometimes a proptosed eye has also some lateral displacement. This cannot be measured by the exophthalmometers available. By measuring the distance from the ipsolateral margin of slide D to the respective stems at the time exophthalmometer readings are

FIG. 3.

made the lateral position of the globe in the orbit is determined, and can be used to assess subsequent lateral deviations. I am indebted for this useful suggestion to Prof. Arnold Sorsby.

The instrument is made by Messrs. Theodore Hamblin Ltd., London.

Summary

A simple device for the direct measurement of exophthalmos is recorded. It is handy and inexpensive. It also allows the measurement of progressive lateral displacement of the globe.

THE TREATMENT OF CONVERGENCE DEFICIENCY, 1933-1944*

BY

MISS SHEILA MAYOU

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1.—Introduction

THE purpose of this paper is to outline the progress made in the treatment of convergence deficiency at the Central London Ophthalmic Hospital during the past eleven years and to set out, in somewhat greater detail, the methods developed by the author which are now employed to treat it.

Patients with eyestrain have been receiving treatment in orthoptic departments for a number of years but since the war their numbers have considerably increased. Some patients can be relieved of their symptoms by the correction of their refractive errors and this may result in a permanent recovery; but if there is a true convergence deficiency, the recovery is likely only to be temporary and there may even be no improvement at all; moreover many patients complaining of eyestrain will be found to have no significant refractive errors.

Eyestrain symptoms may, of course, be due to defective muscle balance, to defective binocular vision or to uncorrected errors of refraction; these causes are not, however, covered in this paper. The cases of convergence deficiency discussed below have had any refractive errors which may have been present corrected before treatment. They have full binocular vision, little or no imbalance —not more than one or two degrees of deviation (either esophoria or exophoria) measured on the Maddox rod test: on the Maddox Wing test they may have a normal reading (0·01) or an exophoric reading of 2△ - 4△ which is said to be physiological: the majority, however, show an exophoria of 6△ - 8△ and some patients show an exophoric reading of 20△ or more. In no case which has been treated have there been any neurological or pathological conditions to which the symptoms could be attributed.

No attempt has been made to cover the treatment of the phorias —exophoria, esophoria and hyperphoria—which undoubtedly cause symptoms similar to those of convergence deficiency but are neither so frequently seen nor capable of such rapid treatment.

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All measurements referred to throughout this paper are in prism dioptres. The relationship between the prism dioptre and a degree of deviation is 7△ prism dioptres = 4° of deviation.

2.—Development of treatment

The increasing importance of convergence deficiency cases and consequently of their treatment is, perhaps, most easily demonstrated by the following table. This is an analysis of the patients who have been seen by the orthoptic department of the Central London Ophthalmic Hospital during the years 1942-1944 classified under three headings, squints, phorias, and convergence deficiency.

TABLE I

Year	Squints	Phorias	Convergence deficiency
1942	124	24	162
1943	261	36	240
1944	214	29	176
	599	89	578

It will be seen that over this period the number of patients suffering from convergence deficiency was approximately the same as the number of squint cases. It must be remembered, of course, that during these war years a large number of children—who normally provide something like 80 per cent. of the squint cases—had been evacuated and that until comparatively recently the L.C,C. clinics have also been closed. On the other hand, there has undoubtedly been a marked improvement in the diagnosis of convergence deficiency and consequently in the number of these cases sent for treatment.

The increased attention which has been paid to the treatment of convergence deficiency and the improvement in the results achieved is shown in the following analysis of cases treated at the Central London Ophthalmic Hospital from 1933-1944.

It will be seen from this table that of the total number of patients treated, no less than 72 per cent. were cured. The patients who showed some improvement only—7 per cent.—had all completed their training and although their tests were normal, they continued to have some symptoms. Sixteen per cent. of the patients, for one reason or another, did not complete their treatment; some of these might have been cured: most of them had shown some improvement; but their treatment, according to the standards mentioned later in this paper, was incomplete and they are therefore not taken into account in assessing the value of the treatment.

TABLE II

Year	Cured	Some Improvement	No Improvement	Treatment Incomplete	Total
1933	1	1	—	—	2
1934	7	—	—	—	7
1935	2	2	—	1	5
1936	2	3	—	—	5
1937	3	1	—	—	4
1938	19	—	1	—	20
1939	3	—	1	—	4
1940	5	1	1	—	7
1941	33	—	2	7	42
1942	75	1	7	15	98
1943	110	12	3	21	146
1944	86	12	8	34	140
Totals	346	33	23	78	480

The failures represent the remaining 5 per cent. and under the circumstances it may not altogether be unreasonable to assume that they were unsuitable for treatment and that their symptoms were due to their general health being at fault rather than to true convergence deficiency.

These figures are confirmed by the results obtained by Miss K. M. Parsons at Chester Royal Infirmary who employs similar methods to those in use at the Central London Ophthalmic Hospital, and by the author's records of private patients. These results are set out at Appendix " A."

A comparison between the totals in Tables I and II for the years 1942-1944 reveals an apparent discrepancy of 194 cases. The figures in Table I, however, represent all cases where the preliminary test confirmed that the patient was suffering from convergence deficiency. Table II includes only those who actually attended for

APPENDIX "A"

1. Convergence deficiency cases seen at Chester Royal Infirmary by Miss K. M. Parsons 1942-44.

	Over 30 years			Under 30 years			Total	Percentage
	1942	1943	1944	1942	1943	1944		
Cured ...	3	12	14	1	7	17	54	93·1
Improved	1	1	—	—	—	1	3	5·2
No better	—	—	—	—	—	1	1	1·7
	4	13	14	1	7	19	58	100

NOTE:—In addition, 14 cases were tested and were found to be suffering from convergence deficiency, but did not attend for treatment.

2. Private patients seen by Miss Sheila Mayou, 1940-44.

	Under 20	21–30	31–40	41–50	51–60	Over 60	Total	Percentage
Cured ...	15	22	18	8	12	6	81	87
Improved	—	1	3	1	—	—	5	5·4
Incomplete	—	1	—	2	1	1	5	5·4
No better	—	—	1	—	1	—	2	2·2
	15	24	22	11	14	7	93	100

treatment. The discrepancy is thus accounted for—as must be expected in a free clinic in war-time—by patients who because of the difficulties of travel or the prior claims of war-work either did not attend for treatment at all or whose attendances were too irregular for any progress to be possible.

The effects of the blitz and of the flying bombs are reflected not only in the number of attendances but also in the amount of time which it was found practicable to devote to the treatment of cases. Thus in 1940 and the early part of 1941, work came almost to a

standstill. In 1942 and 1943, the orthoptic department was working for only three half-day sessions a week: this was increased to five in September, 1943, and in February, 1944, four more were added, making a total of nine sessions a week. The advent of V1 and V2 caused a sharp falling off in attendances as many of the patients were evacuated and did not return to complete their treatment. The number of sessions was accordingly reduced, but the orthoptic department, in spite of one unpleasantly close association with a V2, was always able to handle the number of patients available.

It is worth noting that in 1942, in spite of the war-time conditions the number of patients cured exceeded the combined total of cures obtained in the previous nine years.

The equipment in use during the latter part of the period with which this paper is concerned comprised two synoptophores, one synoptiscope and a myoscope for the treatment of patients at the hospital itself, and a diploscope and stereograms for home exercises.

3.—Comparative methods of treatment

I think most orthoptists employ methods similar to those which we use at the Central London Ophthalmic Hospital, which are described in detail later in this paper. There are, however, minor differences. It is thought that the symptoms caused by close work, which are not due to a muscle imbalance, or any refractive error, are caused by suppression. These patients can be treated by anti suppression exercises with success, and when the area of suppression has been overcome, the binocular abductions and adductions are increased to their normal range of 10 △ and 80 △. Experience has shown, that although the symptoms may have disappeared after six lessons any undue strain or illness may cause them to return. The only safe course, therefore, is to continue until all signs of suppression have gone and this usually takes twelve or more lessons.

When treating a squint, suppression is said to be overcome when diplopia is established. In the same way, when treating a convergence deficiency or an orthophoric emmetropic patient complaining of symptoms, I try to obtain diplopia, voluntary diplopia by converging the eyes, and heteronymous diplopia by looking at a distant object and seeing an intermediate object double. By making the patient aware of diplopia I do not think it necessary to spend valuable time and lessons overcoming the suppression.

An entirely different approach to the problem of convergence deficiency treatment comes from South Africa where Mr. N. A. Stutterheim treats convergence as a kinetic power of the mind

and its insufficiency (asthenovergence) as a kinetic disorder. He maintains*:—

"Asthenovergence is the cause of eyestrain. It also plays a part in ophthalmic migraine and other disorders involving bifoveal vision. Asthenovergence can be cured by methodically calling into action the power of convergence and this is done by kinetic treatment. The power of convergence is present in every normal human brain but in cases of asthenovergence it has not been sufficiently unfolded. Kinetic treatment is certainly not a 'training' and by no means a muscle training. The ocular muscles are in no need of training in cases of eyestrain; they are strong enough without such treatment voluntarily to do many times more work than convergence asks them."

It is not proposed in this paper to do more than touch on the methods which Mr. Stutterheim uses which are set out in detail in his treatise. The key to his treatment, however, is the use of a battery of prisms, which are placed, base out, in front of the patient; in order to overcome the diplopia so caused, the patient is forced to converge and by gradually increasing the strength of the prisms he can be taught to maintain binocular vision over a wide range.

On their first test, most patients complaining of eyestrain cannot overcome more than a 20 △ prism, base out, and many cannot overcome more than 10 △. Convergence power of 50 △ and over should be considered normal, although even with a fairly good range promptness and endurance may be insufficient; training is therefore usually continued until the patient is able to maintain binocular vision with an 84 △ prism base out.

Good results are claimed for these methods which require on average four to five weeks of kinetic treatment given every weekday for ten minutes, before (in Mr. Stutterheim's own words) "the power of convergence is fully unfolded."

At the Central London Ophthalmic Hospital, when the treatment of convergence deficiency first started in 1932, we worked on suppression and increased the range of convergence in order to relieve the symptoms of the few patients we had for treatment. But it soon became apparent from an examination of a number of private patients who had been treated on similar lines that after about two years the symptoms returned.

As a result, we started to teach every patient to converge voluntarily and this has remained the basis of our treatment ever since. To do this, we made the patient look at any object in front of him, for example, a small light, and taught him to converge both eyes equally without effort. This caused homonymous diplopia which

* Eyestrain and Convergence, by N. A. Stutterheim. Published by H. K. Lewis & Co., Ltd., 1937.

he should be able to maintain for a few seconds before letting the two images merge into one. Once the patient knew what was wanted and was able to do it easily, his symptoms disappeared and it was found that very few who had been trained to this standard ever had to return for further treatment. This is the standard we now aim to achieve with all patients and we have proved conclusively that the average person, provided he has normal muscle balance and is prepared to co-operate and concentrate on his treatment, can be taught voluntary convergence in seven half-hour periods and can be freed, probably for ever, from the distressing symptoms which convergence deficiency can cause.

4.—Symptoms and causes

The most usual symptoms of which patients complain are headaches, either during or after working hours, or nausea, giddiness and occasional diplopia: other common symptoms are sore eyes and dry lids—a feeling of " grit " in the eyes, or simply a drawing feeling or acute pain in one or both eyes.

Some patients find themselves unable to read in comfort or to sew for any length of time—often as little as ten minutes being sufficient to start the symptoms; others may be unable to go to a cinema or ride in a car, bus or tram without discomfort. Some attribute the commencement of their symptoms to an illness, to overwork or to general tiredness and in nearly all cases it is found that if the patient is sent for a holiday or ceases to sew or read as the case may be, the symptoms disappear, only to become pronounced again as soon as work is resumed.

A large number of the patients who have been treated during the war have been employed on close work in artificial light. Many were women who, after years of running a home, came back either to part-time or full-time war-work and were often employed on precision work of various kinds or as clerks, typists and on telephone switchboards. Of the men, too, many were clerks, draughtsmen and machinists, working longer hours under more trying conditions than they had previously done.

Some of the most successful cases were young men, members of the A.T.C., who would later be trained as pilots for the R.A.F. or for the Fleet Air Arm under the Navy " Y " Scheme who had been turned down because of convergence deficiency. A number of these patients were treated and every one was subsequently accepted as a pilot when he had completed his treatment. A few service pilots were also seen whose eyes had become unsteady, some as a result of crashes; these, too, were all cured.

Some convergence deficiencies are of traumatic origin. In these cases it is not necessary for the patient to have been unconscious, since any severe blow to the head may be sufficient to cause the

symptoms which can be relieved by exercises. A frontal sinus that has had surgical treatment may give similar symptoms, but these cases are usually phorias and are treated as such.

There have been a few patients with migraine. Some of these have been completely freed from further attacks as the result of treatment; others have improved in that their attacks have neither been so frequent nor so severe; few have shown no improvement. It seems clear, therefore, that a migraine patient who shows ocular symptoms can at least gain some alleviation from convergence deficiency treatment.

A type of patient who is likely to be seen more frequently in the near future is the man who has returned to civilian life after many years of active service during which his opportunities for reading and concentrating on close objects for any length of time will often have been limited. Now that he has a desk job he finds himself unable to work in comfort for more than short periods at a time.

On the other hand, the symptoms described above are not the only guide to the necessity for convergence deficiency treatment. Cases have been seen where convergence has been very poor, one eye diverging to an object held as far away as eight inches, who have not complained of any symptoms at all. These cases usually have an exophoric reading on the Maddox wing test and are sometimes referred for treatment as it is thought that their symptoms might develop in later life.

Generally speaking, patients with large exophoric readings should be given treatment whether or not they complain of symptoms, since these are liable to develop later. Patients with a low exophoric reading, however, are not treated unless symptoms are

TABLE III

Age in years	Percentage of cases
Under 20 ...	17·7
21–30	37·7
31–40	26·0
41–50	10·7
51–60	5·0
Over 60... ...	1·3
No record ...	1·7

already present, although they may sometimes be given simple exercises to practise at home by a surgeon, without being referred to an orthoptic department.

It seems clear that the age of the patient has little bearing on the question of convergence deficiency. This is shown by Table III, below, which covers *all* cases tested or treated at the Central London Ophthalmic Hospital during the period 1933-1944.

It will be seen that the vast majority of the patients were under forty. Perhaps too strong an inference should not be drawn from this, since it is arguable that older people have less inclination to bother with a series of attendances at a hospital. Nevertheless it is evident that convergence deficiency can be diagnosed and cured irrespective of the age of the patient.

5.—The psychological approach to treatment

This may sound a long-winded description of a simple process; but the fact remains—as is so often the case—that the patient's attitude towards treatment is all-important and may make the whole difference between success and failure. One of the essential factors in helping a patient who suffers from convergence deficiency is to explain in simple terms exactly what the trouble is and to impress upon him that a quick and permanent remedy lies largely in his own hands.

The first thing we do with a convergence deficiency is to explain exactly what the treatment entails; attendance at the Hospital for one half-hour's treatment each week, probably for six weeks, coupled with exercises at home for at least ten minutes every day. The patient is warned to expect his symptoms to be rather more pronounced during the first three treatments but is told that he may expect a definite improvement to begin after the third or fourth lesson. Most patients can find time for these lessons, particularly when they have been suffering for a long time and are really uncomfortable; the more intelligent can often manage in less than seven attendances once they understand exactly what is required of them and concentrate on their home exercises.

What many patients cannot understand is how these severe and unpleasant symptoms which may have been troubling them for many years can be overcome in so short a time and this distrust frequently makes them unwilling to go through with the treatment. In order to overcome this reluctance, we explain that although the muscles of the eyes are normal, the sight is normal and the ability to use both eyes together is normal, the power to converge or focus both eyes together on a close object is not sufficiently developed or needs too much effort; that this inability to converge or the undue effort required is the cause of the symptoms which the patient, unbeknown to himself, has been aggravating whenever

he has drawn his eyes away from a close object instead of forcing them to look at it and maintain convergence; hence his reluctance to read or do more close work than necessary. If, however, he can be taught to converge his eyes without effort his symptoms will disappear, but that during the early stages of treatment while his eyes are learning to converge, he must expect a recurrence of the headaches and other symptoms from which he has previously suffered whenever he attempted to focus on close objects.

Anyone who tests or teaches convergence daily, as we do, is all too apt to take for granted that the patient understands what is wanted, but experience has shown that it is impossible to be too careful in explaining what is required. Many patients will look at a near object half-heartedly because it hurts them to do so or because they imagine that that is all they are asked to do. But if some definite object, such as the nib of a fountain pen, is held about six to eight inches in front of the patient's eyes and he is told "now look at the nib; make it into one object and see it clearly," he will, if he possibly can, converge both eyes in order to look at it. On being forced to converge, he will probably draw his head back from the pen nib, thus showing his dislike of looking at a close object; it can then be explained to him that this discomfort is due to his lack of convergence, or, where he complains of diplopia, to his inability to maintain convergence. In this way he can be made aware, at the start of his treatment, of the effort which has been required whenever he has attempted to focus on any close object and he begins to have a practical understanding of the cause of his symptoms.

6.—Detailed treatment

All patients who are referred to the Central London Ophthalmic Hospital for convergence treatment, whether through an out-patient department or a private practice, have been carefully refracted. Those who have a refractive error are normally given glasses, but where the error is small, it is often better to see the result of the exercises before the glasses are ordered as they may prove to be unnecessary. In some cases, however, even after treatment, the patient may feel more rested if he wears his reading glasses.

(*a*) *Preliminary test*:—On arrival at the orthoptic department, the patient's detailed history is taken and the movements of the eyes are tested in the nine fields to see that they are normal. The visual acuity of each eye is tested and if glasses are worn constantly it is tested with and without correction. Visual acuity is usually good but sometimes one eye will read one line more than the other and occasionally amblyopic eyes are found with 6/36 vision; these amblyopic cases are more difficult to train because, although

their symptoms can be relieved, it is impossible to teach voluntary convergence.

The next step is to test convergence. The patient is first of all asked to look at the point of a pencil or pen held about two feet from his eyes and is told to keep looking at the point as it is brought gradually closer to his face. If he can do this until the point is within two inches of the bridge of his nose, the test can be regarded as satisfactory.

The second convergence test is to hold the pencil point about three inches from the bridge of the patient's nose and tell him to look at it; he should, with little difficulty, be able to converge both eyes and see the point as one fairly clear object.

Most patients find that they are able to pass the first test of *gradual convergence* unless they have a high exophoric reading on the Maddox Wing test but a great many will, to start with, fail on the second test of *jump convergence*. These pencil tests can be done more accurately on the Livingston gauge which was specially designed with these tests in view.

If, however, the patient can manage both tests, he is made to look at a small object or light about six feet in front of him. By converging both eyes—by "going cross-eyed"—he should be able to double the light and keep it double for about thirty seconds.

One of three things may happen, and it is most important that the patient's eyes are observed:—

1. Most frequently he will look at the light without any idea as to how to double it.

2. He may be able to double the light but not to maintain it for more than a moment.

3. One eye only may converge to get the diplopia; in this case the symptoms will usually be found to be of a monocular type.

If, however, the patient can double the light by converging both eyes equally and can maintain the diplopia steadily for about thirty seconds, convergence exercises will not be necessary.

The results of the remaining tests are as follows:—On the Worth four-light test, the patient will see four lights. On the Maddox Rod test, he will usually be found to be orthophoric or to have an esophoric or exophoric deviation of not more than two degrees. On the Maddox Wing test, there may be any one of the following readings: the arrow may point to 0·01, showing orthophoria but more usually will be between 2 $\triangle$ – 4 $\triangle$ exophoria gradually moving to 8 $\triangle$. Some patients, however, will see it at as much as 16 $\triangle$ – 22 $\triangle$ exophoria.

On the synoptophore or synoptiscope, he may have all grades of binocular vision—simultaneous perception, fusion and stereoscopic vision; simultaneous perception slides (which have no fusion

element) will show some suppression and his answers on the stereoscopic slides may be slow or slightly inaccurate; his fusional adduction will be below normal (few patients can adduct to 60 △) and he will have no adduction or voluntary convergence on simultaneous perception slides.

If the adduction is approximately 60 △ to 70 △ it may be considered good and this range of adduction or fusional convergence can be reached by gradually converging both tubes of the synoptophore until the pictures on the fusion slide break. Once they have come apart, it should be possible for the patient to make the effort of " jump convergence " and fuse the two slides together at the convergent angle. This, at first, proves to be too difficult for the average patient but comes easily with practice.

If, however, the patient can manage this, the fusion slide is replaced by a simultaneous perception slide. With the tubes set at a convergent angle of 50 △, a patient with good convergence and normal control should be able to bring the ball into the square and hold it in the centre for a few moments.

Obviously, few patients are able to do this at first and if they can, convergence exercises are unnecessary.

(*b*) *Home Exercises*:—Immediately following his preliminary test, the patient is taught how to converge and is given exercises to practise at home.

In the first of these home exercises the patient is told to hold a pen about twelve to eighteen inches from the bridge of his nose and to look at it with both eyes; he must then bring the pen closer to his nose, concentrating on it with both eyes all the time, until it is within two inches of his nose. This is similar to the gradual convergence test mentioned in para. 6(a).

When he can manage the first home exercise, the patient can go on to the second. In this, he has to hold the pen about six to eight inches from the bridge of his nose and in a direct line with a small light or torch which has been placed two or three yards away. If he looks at the light, the pen will be double due to physiological diplopia; when he looks at the pen his eyes are converged so that the light is double (homonymous diplopia). The object of this exercise is to keep the eyes in a convergent position without fusion (*i.e.*, to converge the eyes by using the adductor muscles); the patient is therefore told to see the light double by looking at the pen and then, having removed the pen, to attempt to continue to see two lights. It is important that when the pen is removed he should continue to look at the place where it was held and not on any account look at the light. As long as the eyes stay converged, the light will remain double but immediately they are relaxed the two lights will merge into one. The patient is told to practise for about ten minutes daily throughout the period of

treatment, until by the end of treatment he should be able to look at any object, distant or near, and without any effort, converge both eyes so as to see the object double. This is known as *voluntary diplopia*.

(*c*) *Lesson one* :—The patient spends only the first few minutes of this lesson on anti-suppression exercises and then goes on to fusion slides. Here, with the aid of minus lenses, he will endeavour to increase his range of convergence and at this stage it is usually best to leave him to work by himself; provided that he really understands exactly what he is trying to do, the mere fact that he is operating the tubes himself and can feel his hands moving the slides helps him to make his eyes follow the pictures. By keeping the slides fused together, he is forcing his eyes to converge and as soon as the slides come apart he moves the tubes back until they fuse again. In this way the patient can teach himself to keep the pictures fused to 60 △ or 80 △ of convergence.

(*d*) *Lesson two* :—The patient starts off with the exercises which he practised in lesson one, but after a few minutes with the minus lenses he should be able to converge to 70 △ with ease. The lenses are therefore removed, and he spends the rest of this lesson attempting to achieve the same standard without their aid; by the end of this lesson, most patients are able to attain 70 △ of convergence unaided, without difficulty.

(*e*) *Lesson three* :—Continuing the process of refresher training, the patient spends the first few minutes on anti-suppression exercises, followed by convergence with fusion slides. Provided he has achieved the standards set in lesson three, he then proceeds to "jump convergence" exercises. For these, the tubes are set at a convergent angle of 30 △ so that the pictures appear side by side, and by converging his eyes, the patient should be able to fuse them; when he can do this at 30 △, the convergent angle is increased to 40 △. It is important at this stage that the patient should be allowed to *look away* from the synoptophore between each attempt to look in and fuse the two pictures.

When the convergent angle is set above 40 △, the younger patients frequently say that the slides, when fused, appear smaller and somewhat blurred: presbyopes, on the other hand, can retain a clear image for a considerable time, varying with the degree of presbyopia.

To encourage convergence to 60 △ the patient should be told to make the pictures go out of focus and look in front of them rather than at them; this will help him to fuse the pictures together. He should be able to do this at a convergent angle of 60 △ and maintain fusion while the synoptophore tubes are locked at this angle and moved from side to side.

The next step is to teach the patient to relax his eyes and

"break coincidence" *without* moving his head away from the synoptophore. Most patients find that once the pictures are fused at the convergent angle they are unable to relax sufficiently to let the fusion go so as to resolve the image into two pictures. This must be practised until it is effortless.

An alternative exercise to stimulate convergence and relaxation is to set the tubes containing the fusion slides at zero so that the patient sees the two pictures fused together; by making a voluntary effort of convergence, he should be able to break the fusion and see the pictures side by side; when he relaxes, they will fuse again. This exercise, however, is more difficult than the jump convergence exercise and should not be undertaken until the patient has mastered gradual and "jump convergence" and is making good progress with the home exercises.

During lesson three, the patient will start to use the myoscope or myoculator. This machine is not essential for the treatment of convergence deficiency but it is a great asset, particularly in a busy clinic. It consists simply of a light projected through rotating prisms on to the wall and a small motor which propels the light across the wall at a variable pace; the motor can be set to move the light horizontally, vertically, diagonally or in a circular movement.

For convergence deficiency cases, the myoscope or myoculator is used in two stages to amplify the training which the patient has done on his own in the two home exercises previously mentioned. The motor is set to produce a horizontal movement of the light at a slow pace and for the first stage the patient is told to hold his finger close to his face and keep his finger in line with the light as it travels across the wall. By looking at his finger, he will see the projected light double and should try to maintain the two lights over the whole range of movement. When he can do this, the patient goes on to the second stage in which he attempts to achieve exactly the same result—the doubling of the light—but without using his finger to help him, by keeping his eyes converged to the arc where the finger was travelling.

Almost invariably, when patients first use the myoscope they try to move their heads or their bodies instead of their eyes and as soon as the finger is removed they focus on the light which becomes single; this tendency needs careful checking; only regular practise at the home exercises and sufficient encouragement during this and the succeeding lessons will enable the patient to master this exercise; but although, at times, he may feel that he will never be able to manage it, success will come suddenly.

(*f*) *Lesson four*:—A quick run-through the exercises of the previous lessons usually reveals that further time must be spent on "jump convergence," relaxation, converging from zero and the myoscope exercises; these are accordingly continued.

When, however, they have been mastered, the patient goes on to the *voluntary convergence* exercise. For this, the fusion slides in the synoptophore are replaced by simultaneous perception slides. These are slides which have no fusion element but have two dissimilar images such as a ball and a square. If the patient has good convergence and is not relying on his fusion power to hold it, he will, by converging both eyes, be able to pull the ball into the square at a convergent angle between 10° and 35°. At first, he may be able to move the ball towards the square or even put it in but will not have the power to keep it steadily in the centre for any length of time. This, however, comes quickly with practice.

By the end of this lesson, the patient should also be able to follow the myoscope light in all directions, making it appear double or single at will, without effort.

By now, too, he should be able to converge or relax on a stereoscope card without the use of the stereoscope, so that he can see the different formations of the letters if the card is simply held up in front of him. This is a simple exercise and should also be continued at home. All that is required is a postcard with two letters, F and L, about ½in. in height and the same distance apart; alternatively, any simple stereoscope card can be used. To relax, the patient first looks at the card and sees F and L; then, by looking right through the card into the distance, his eyes are slightly diverged from the card and he sees the letters as FEL. To converge, he fuses a near object held between his eyes and the card and as his eyes are too convergent to the card he once again sees the letters as FEL; the near object is then removed and the patient should be able to maintain the convergence, until eventually he should be able to converge without the help of the near object. This converging and relaxing—FEL relaxed, FL normal, and FEL converged—helps to give a general tone up to the abductor and adductor muscles.

This exercise can also be practised on the diploscope. The patient will aim to change from the normal DOG in three holes to DOOG in four holes relaxed and to OGDO in four holes converged and should be able to manage each position without difficulty by the time treatment is complete. The diploscope is, however, to some extent a luxury with hospital patients and it is only used in the clinic and not for home exercises.

(*g*) *Lesson five*:—This is spent on a general work-through of all the exercises which the patient has learned. Some patients will not yet have achieved the necessary standard on one or more of the exercises and may not have covered the full syllabus of the previous lessons; others will spend this lesson on a general tone up; a few will be able to dispense with further exercises altogether. The

average patient will still need some further treatment, however, and particular attention is paid during this lesson to the voluntary convergence exercises since they contribute most of all to accuracy of convergence.

(*h*) *Final test*:—In the preliminary test, a careful enquiry was made into the symptoms noted in each case and it is usually found that these symptoms disappeared as soon as voluntary convergence was established.

The patient is now ready for the final test which comprises a series of routine checks. Convergence should by now be effortless and voluntary convergence should enable the patient to double any small image which is at least a yard away and to hold the two images for a short time without difficulty. The test for visual acuity will show improved vision—possibly as much as a line better—due to the fact that suppression has been overcome even though comparatively little time has been spent on anti-suppression exercises. The Worth four lights are seen as four unchanged; the Maddox Rod measurement is usually orthophoric or shows exophoric or esophoric deviation of 1 Δ which has probably remained unchanged. There may be little or no alteration in the deviation on the Maddox Wing test; if the patient had an exophoria of 4Δ – 6Δ before treatment, this will probably measure exophoria 2 Δ – 4 Δ or may be unchanged after treatment. If, however, his deviation was 14 Δ – 16 Δ, this may vary after treatment anywhere between 2° – 16°; these different results do not, however, seem to matter, and some patients do come down to normal. Synoptophore tests should show no deviation and little or no suppression with simultaneous perception slides. Voluntary convergence with simultaneous perception slides should be good, as should gradual and " jump convergence " to 80Δ with relaxation; lastly, the patient should have good abduction power to 10Δ and full stereoscopic vision.

Provided he can pass all these tests and is free from symptoms, the patient is ready for discharge. Careful supervision is necessary, however, throughout the period of treatment as a few cases become esophoric and may start convergent spasms which are difficult to overcome. This should not occur if sufficient attention is paid to relaxation exercises and it is therefore essential that patients suffering from convergence deficiency should be under constant supervision and that, once convergence is established, part of each lesson should be spent on relaxation.

7.—Conclusions

From the author's experience of convergence deficiency cases over the past eleven years, it is suggested that the following general conclusions may be drawn :—

(*a*) The number of patients of all ages suffering from convergence deficiency is large and the symptoms, which may include migraine, extremely unpleasant; the psychological element is often present and particular attention must be paid to the patient's mental approach both before and during treatment since the successful remedy lies largely in his own hands.

(*b*) Care should be taken when testing and treating a patient with an esophoria since many of these have a convergence deficiency as well.

(*c*) Even patients who are orthophoric and can actually converge may benefit from treatment, while many patients with small refractive errors are able to dispense with their correction after they have been treated.

(*d*) Above all, treatment can be quick and effective—on average, two tests and five half-hour lessons—but must be taken under trained supervision; it should aim at building up voluntary convergence and teaching the patient to relax once convergence is established, since voluntary convergence alone can prevent a recurrence of the symptoms in after years. If a patient is discharged as cured after undergoing the treatment described in this paper, it may be said with some certainty that with the exception of the patient with migraine symptoms who may need a subsequent refresher course, the risk of his having to return for further treatment is infinitesimal.

THE ULTRA-VIOLET OPHTHALMOSCOPE*

BY

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THE ophthalmoscope has long been used for observing the interior of the eye, and thus determining the appearance of the media, the condition of the retina, choroid, and optic nerve, and for a variety of other conditions of this organ. As is well known, the present-day ophthalmoscope relies upon the use of white light and ordinary vision, though in past years a number of modifications have been introduced into the design and mode of operation of this instrument.

The fluorescence of the eyes of humans and lower organisms has been known since the earliest days of fluoro-chemistry, though the first accurate description of this phenomenon was probably

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that of Helmholtz[1] in 1896. The fluorescence of the eyes of lower animals was described later by Kohler[2] and by Hess[3].

In more recent years the fluorescence of the eye has been widely mentioned by writers in a number of different connections with fluoro-chemistry[4]. The fluorescence of the eye has assumed importance lately with the introduction of the uranine tests, such as those for circulation[5] and for death[6].

In studies on the use of uranine injections for distinguishing real and apparent death the writer has observed the interior eye structure of living animals such as humans, cats, dogs, and rabbits to have a bright green luminescence with filtered 3650A ultra-violet light (*e.g.*, radiation from a G.E. H-4 lamp). In an investigation of several hundred living and dead animals, however, it has been observed that after sudden death this bright green emission may change to a dull blue.

In previous studies, to my knowledge, where the fluorescence of the eye was being observed a beam of ultra-violet light was merely directed into the orbit and the radiation-response of the various structures noted qualitatively. In my more recent studies on the fluorescence of the interior structures of the eye, I have designed an ophthalmoscope which employs filtered ultra-violet light instead of visible light. It possesses certain other features which may make it of general interest to those engaged in the experimental study of ophthalmology and related fields. A description of the *ultra-violet ophthalmoscope* follows:

The device consists of a round mirror of silvered glass or unpolished aluminium, with a small perforation in the centre. The surface of the mirror can be either concave or plane, or, the mirror in a more improved form can be reversible, one side of which is plane and the other concave. Mounted in the central perforation of the mirror is a small circular window of an ultra-violet opaque medium, such as Noviol glass or Wratten 2-A filter. Behind this on a rotor may be mounted one or more filters of transmission suitable for special observations, such as colour analysis by filter radiometry.

The ultra-violet ophthalmoscope is used exactly like its conventional counterpart. A source of filtered ultra-violet light (of "long wavelength," *i.e.*, 3650A) is placed slightly behind the plane of the subject's face, and preferably on the same side as the eye to be examined. The filtered ultra-violet light beam is directed toward the reflecting mirror which is held in the line of vision of the subject and directed into the eye: observation is made through the centre aperture, the presence of the ultra-violet absorbing glass in the aperture preventing ultra-violet light from passing into the observer's eye, exciting fluorescence, and so creating a masking effect. A lens (double convex) of pyrex or quartz may be placed

at the appropriate distance between the ophthalmoscope and the eye under inspection if greater resolution is desired. The lens must not fluoresce, and all examinations are made in a darkened room.

There appear to be a number of interesting possibilities for the ultra-violet ophthalmoscope in medical and biological science, not only for the study of the internal eye structures, but also for the examination of such exposed regions as the brightly fluorescent sclera. As is well known, cataract tissue fluoresces differently than its normal surrounding tissue: hence, with this instrument greater assurance might be had of complete removal of all pathological tissue from the cornea; lesions of the cornea and sclera are revealed by their green fluorescence after uranine solution has been dropped into the eye; neoplasms and other disease tissue present marked differences under high intensity ultra-violet light, as frequently do the different kinds of normal tissue.

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MOSCOW EYE HOSPITAL*

BY

A. USPENSKY

ON Gorky Street, Moscow's main thoroughfare, stands the Moscow Eye Hospital, one of the largest and oldest ophthalmological institutions in the country.

Founded 120 years ago its scope was at first quite limited. There were only two staff doctors, only 30 people a day passed through the out-patients' department and the hospital was equipped with only 20 beds. Now the out-patients' department is visited daily by 500 people and the number of beds has increased to 250.

The growth of the hospital dates from the post-revolutionary years when the whole of the medical service was re-organized. Thus, for example, in 1917 only 200 clinical analyses per month

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were made at the hospital whereas by 1944 this number had increased to 5,000.

When a patient attends the hospital in connection with some eye trouble he is examined not only by an oculist, but in case of necessity by doctors specializing in therapy, neuro-pathology, skin and venereal diseases, stomatology, pediatry, etc. The hospital has every kind of laboratory and X-ray department attached to it.

Treatment at the hospital and in the out-patients' department, as well as all necessary analyses are free for every citizen. Operations are also carried out without charge. An average of 250 operations a month are performed at the hospital.

The Moscow Eye Hospital is not only a medical institution, it is also an important clinic. It maintains close contact with the Academy of Sciences of the U.S.S.R., the Academy of Medical Sciences, and related medical institutes (in particular the Institute of the Brain, the Institute of Neurology and others); it holds periodic and regular scientific conferences for exchanges of experience designed to help ophthalmologists. The hospital has trained 2,000 ophthalmologists.

The activities of the Moscow Eye Hospital extend beyond the borders of Moscow. Through its methodological department it renders regular assistance and consultation in difficult cases to doctors working outside Moscow.

The hospital's regular consultant is Professor Strakhov who has been working at the hospital for about 50 years. Such distinguished ophthalmologists as Filatov, Golovin, Odinstov, and others, have also worked in the hospital.

During the war the Moscow Eye Hospital treated thousands of sick and wounded soldiers and performed more than 10,000 operations. Eighty-two per cent. of the wounded and 88 per cent. of the sick were returned to the ranks for further service.

At present the Moscow Eye Hospital is preparing to mark its 120th anniversary.

ANNOTATIONS

A National Eye Service

At the time of writing the National Health Service Bill is rapidly going through its Parliamentary stages, and, for better or for worse, great and far reaching changes are taking shape in the practice of medicine in this country. In these changes ophthalmology will share, and it is essential that all ophthalmologists—particularly those of the younger generation—should carefully consider their implications. Most thinking people are agreed that the medical services in

this country are by no means perfect and that in large areas adequate facilities, especially in the specialist services, are not as readily available to the ordinary people as could be wished. Politically all parties in Parliament are committed to their provision as a national charge; but whether the present Bill provides the best mechanism for their distribution is a matter which some will question. Certainly the proposals are revolutionary and drastic; no one can complain that courage has been lacking in their formulation. Whatever our individual ideas regarding them may be, medicine—and ophthalmology—is entering upon a new epoch.

At the present moment the Bill has passed its second reading in the Commons, and it may therefore be assumed that the general principles it contains will become law. Amendments will doubtless be made in its further course, and much of the detail requires to be filled in by regulations; but we now have before us the main trend of policy which, in due course, it will be open to each member of the profession to implement or reject. At a later stage, when further details have emerged, we will return to the subjects in these pages.

Under the provisions of the Bill ophthalmology is (rightly) included in the Hospital and Specialist Services and it is to be conducted ultimately in special ophthalmic departments and clinics forming part of these services. The whole practice of ophthalmology is therefore to be institutionalized so far as the National Service is concerned. All the hospitals in the country, voluntary and municipal, become the property of the Minister, and apart from the teachiug hospitals for which independent and special provision is made through Boards of Governors, their administration is to be entrusted to some 16 or 20 Regional Hospital Boards composed of persons chosen by the Minister (after consultation with the regional medical school, medical representatives, local authorities and those with experience of voluntary hospital management) for their individual suitability for the task. These bodies under the Minister will hold the key positions in the medicine of the future. The country is thus to be regionalized, each region being associated with a university medical school, and the Regional Boards, with the help of Local Hospital Management Committees appointed by them for the day-to-day administration of individual large hospitals or related groups of hospitals, will be responsible for the administration of a co-ordinated hospital and specialist service for the various regions. For their general finance they will look to the Exchequer.

Ophthalmologists participating in the National Service will therefore be appointed by and in the employment of the Regional Boards (or the Boards of Governors of Teaching Hospitals): they may be whole-time on the staffs of hospitals or part-time continuing any private practice outside the service as may be available. If—and only if—they are part-time, they will be able to avail themselves of

pay-bed accommodation in hospitals for private patients, provided the Minister decides that such beds are not required for the general service and provided the fees charged are within a maximum to be fixed by him. In so far as the National Service is concerned he will see and treat patients referred for ophthalmic consultation by practitioners in the same way as in other specialties.

The widespread calls for routine sight-testing and the provision of spectacles, however, necessitate more elaborate arrangements, and for this purpose it is proposed to set up ophthalmic clinics in the hospitals and in association with them. These will be staffed by an ophthalmic specialist in charge, junior ophthalmologists under him (in the larger clinics), and, also under him, optician-refractionists who will do the bulk of the refraction work. There is also provision for orthoptists. Spectacles will be obtainable either at the clinics themselves or at the premises of dispensing opticians taking part in the service. The whole of this service is "free," although if spectacles have to be prematurely repaired or replaced as a result of carelessness, and if the patient chooses to be supplied with more expensive articles than those normally supplied, he will be expected to meet the additional cost involved.

It is the belief of the Ministry that the resources at present available, particularly in clinic accommodation, will not allow of the universal application of such a service when the scheme is inaugurated on January 1, 1948, and to tide over the interval a Supplementary Eye Service is to be inaugurated which essentially continues the working of the National Health Insurance benefit as it is to-day. After a patient gets a recommendation for an eye examination from his practitioner, he may go either to an ophthalmologist or a sight-testing optician of his choice. The difference from the present system is that sight-testing by either of these and the supply of spectacles are "free." These arrangements are under the control of Executive Councils (the bodies which control the general practitioner services of the area) who will entrust the arrangements to specially formed Ophthalmic Services Committees. The ophthalmologist in this Supplementary Eye Service will be employed by and under the disciplinary control of these bodies, as are general practitioners, dentists, opticians, chemists and others, and the remuneration of those participating will be the subject of negotiations. When the Bill becomes law it is the intention of the Minister to undertake an ophthalmological survey of the country, and as soon as he is satisfied that adequate clinic services are available in any area, he may wind up the Supplementary Service in that area. Thereupon all patients desiring eye examination through the National Service will go to clinics only and the ophthalmologist will require to obtain employment through the Regional Boards in the Hospital and Specialist Service.

It is obvious that the widespread (and perhaps ultimately the universal) application of such a scheme will radically alter the habits of all ophthalmologists who take part in it. From the professional point of view the greatest change is our new relationship with opticians. It is certainly true that in the past our relationships as a body with sight-testing opticians have not been the most cordial; but there is every reason to expect that, when each body dove-tails into a co-ordinated place wherein each plays an agreed and appropriate role, the animosities of the past will disappear and give place to co-operation in the future. There are manifestly insufficient ophthalmologists to undertake the total refraction work which a universally "free" service will entail; nor indeed would it be economic that ten years of training (the time which will probably be demanded for the total training of a specialist) should be required for the routine measurement of the optical state of all eyes, a great proportion of which will be "normal." So long as the optical examinations are under adequate medical control, all our requirements should be met, and in this respect this country will provide a more adequate optical service than any other. There is, of course, no reason why the ophthalmologist in charge of the clinic should not himself undertake what refractions he chooses; for the junior particularly this will be a necessity. Moreover, there is no doubt that the running of such a service covering the whole community will keep the available ophthalmologists sufficiently busy.

The present reaction of the individual ophthalmologist to the scheme will certainly vary; and it will doubtless vary still more as succeeding regulations clarify the position with the passage of time. Economic considerations must be postponed until negotiations take place between the Minister and the profession as a whole; but on general principles some may be attracted by the security of a regular pay-packet followed by a pension, even although it may be alterable by any Government of the day, while others may prefer the adventure and opportunities of free practice. From the professional point of view everyone will probably agree with the regionalization and organization of specialist services; some may find relief in the regularized routine and regimentation which even the mildest state service must entail; others may temperamentally abhor its restrictions; and those who have lived for long in the atmosphere of a voluntary hospital will, in many cases at any rate, be regretful. Those who wish to make the best of both worlds may have some anxiety regarding the vast powers vested in the Minister (whoever he may happen for political reasons to be), and fear that his complete over-riding control of the hospitals—without which consulting practice is impossible—puts specialists and consultants too much in his power. The fear may be accentuated by the authoritative statement in the House of Commons that it was the

policy of the Labour Party to have a full-time state salaried service "when the fruit was ripe." From the point of view of the people —and that in the end is the important matter—much will depend on the spirit with which the service is worked both by the Government and the profession. There may be a loss in local interest and voluntary effort or even in some cases of personal intimacy and interest. There may be a gain in the technical machinery and financial resources available to medicine—if Treasury control is kind and the Government is inclined (or indeed is able) to implement its growing obligations indefinitely. But in the end the most important factor will be the type of doctor who enters the Service. The present generation will, if they accept it, see it through creditably. But as for the future, only if medicine, in competition with other professions which are as yet free from State control, can continue to attract brilliant young men and reward them sufficiently, and only if they have opportunity for initiative and the full expression of their personalities, will medicine in this country be able to keep up the traditions of the past and the new medical service bring ultimate benefit to the people.

Myopia in Lower Animals

Some of our readers may remember a leading article in *The Times* newspaper at the beginning of this year entitled "Spectacles on nose," and some letters to the Editor on this subject published in subsequent issues. An Exeter rector wrote to point out that in an inventory of the effects of Bishop Walter de Stapledon, at so early a date as 1326 A.D., is noted a pair of spectacles valued at 2/. Such a sum in those days would probably approximate the cost of the best tortoiseshell frames fitted with toric lenses at the present time. In a later part of his letter he quoted a report from *The Manchester Sporting Chronicle* of January, 1888, of a myopic horse having been fitted with concave glasses No. 7, with remarkable results as to its behaviour.

It would be interesting to know whether our Manchester correspondent is able to throw any light on this case of myopia in a horse. To the best of our recollection the refraction of a number of the lower animals was investigated by William Lang and Sir James Barrett in the eighties of last century and their conclusions published in the Moorfields' Reports. The prevailing optical condition found was hypermetropia. We see no reason why a horse should not be myopic, but we never were asked to give an opinion on such an animal.

Many of the lower animals rely quite as much on the sense of smell as on that of sight. The Manchester animal lost all sense

of timidity when being driven with its glasses attached according to this report, but we should have thought that blinkers would have acted just as well, and most horses when we were young were always driven in blinkers.

Charles Kingsley, in *The Water Babies*, asserted that all dragon flies are very short sighted; but as the eye of the dragon fly, we believe, is a compound optical instrument like that of the blue bottle and common house fly, we must assume that he was writing with his tongue in his cheek, as indeed is apparent throughout much of that fairy tale. A dragon fly in spectacles would be a very odd sight to meet during a country walk. One's veracity might well be questioned had one the temerity to say that one had seen such a sight.

ABSTRACTS

MISCELLANEOUS

(1) **Garden, R. Ramsay (Bristol).—The blind child.** *The Practitioner*, p. 180, September, 1945.

(1) In spite of the steady decrease in the number of those whose blindness dates from infancy there is still a considerable group whose sight is so seriously impaired by congenital, developmental and other defects arising during childhood that they have to be educated and taught the art of living by special methods. The object of this article is to give those not familiar with the care of the blind child some information with which they can encourage the distressed parents and to indicate the voluntary and official organizations which deal with the care and education of these unfortunate children.

The first national school for the blind was founded in Paris in 1784 by Haüy. In 1791 Liverpool opened the first institution of the kind in this country, followed soon after by Bristol, Edinburgh and London (at Southwark). Haüy was the first to emboss paper with raised characters to provide reading matter for the blind, while his pupil Braille published in 1829 the six-point system which bears his name.

In Great Britain the majority of schools for the blind are managed by voluntary associations, but the Education Authorities make the fullest use of their organizations, accepting financial responsibility for maintaining most of the pupils. This also applies to higher

education, secondary and technical, and to the workshop training of those leaving school.

The subject is discussed under three headings:—the blind infant, schools for the blind and the partially sighted child.

The blind infant. Although the education of blind children under the age of five years is not compulsory, local Education Authorities have power to provide it from the age of two years. However, the best place for an otherwise normal blind child is at home provided the conditions there are adequate and the mother sympathetic and sensible. This is not the case when the parents are ignorant or ill-housed; then the infant should be admitted to one of the Sunshine Homes maintained by the National Institute for the Blind. Here the aim is to stimulate self-reliance by excursions in the grounds, to give instruction in dressing, in caring for toys and games and in the elements of Braille reading and writing. Behind the blind eyes is the perceiving brain which with agile fingers, nimble legs and alert little noses acquires habit, forms personality and develops imagination.

Schools for the blind. Once school age is reached education is carried on in one of the schools provided by voluntary organizations or by the Ministry of Education. It is now compulsory for pupils who have no sight to be educated in a boarding school. Numerous forms of manual work are taught. Secondary education is provided for blind boys at Worcester College and for blind girls at Chorley Wood. The Royal Normal College admits pupils from the age of five to twenty-one, giving special attention to the teaching of music and stenography besides providing a sound general education. For most of the blind children who do not go to secondary schools courses of training in some form of manual work are provided at workshops recognized by the Ministry of Education.

The partially sighted child. In a number of blind schools classes for the partially sighted have been established. At the Royal School for the Blind, near Bristol, with which the author is associated all the pupils get practice in using Braille up to the age of eleven, and the partially sighted can be taught by this method should the condition of the sight require it at a later age. Also, if it is desirable that the eyes of a myope should be rested for a period it is easy to arrange for education to be carried on by oral methods, with or without Braille. It is desirable to let the educationally blind make use of any sight they may have (and some have a considerable amount) so long as no harm is likely to occur. To those and to the few whose sight shows sufficient improvement whilst at school, partially sighted methods in part or as a whole are readily available. By assistance to their blind companions the partially sighted acquire some character training unconsciously.

A. F. MACCALLAN.

(2) **Diez, Magin A. (Buenos Aires).—Ophthalmological syndrome in frontal lobe tumours.** *Arch. de Oftal. de Buenos Aires*, July-August, 1945.

(2) Ten cases of frontal lobe tumours were studied. Time of evolution from beginning of subjective symptoms ranged from three months to nine years. Average time was six months to two years. Apart from headaches, the initial symptom was diminution of visual acuity, sometimes taking the form of transient amblyopia. Other symptoms were pain in the globe and optic hallucinations.

The ocular signs were the following:—1. Moderate exophthalmos, generally bilateral. 2. Widening of palpebral fissures. 3. Nystagmus on looking at extreme limits, more marked on the side of tumour. 4. Hypertension of central retinal artery. Highest readings correspond to same side as tumour. 5. Bilateral papilloedema; oedema more marked on same side as the tumour. Foster Kennedy's syndrome was not encountered. 6. Visual acuity is diminished on the same side as the tumour. It is generally unimpaired in meningiomas unless they are of long standing and compressing the visual pathways or venous trunks. 7. Visual field changes.

(*a*) Generally the blind spot is of greater size on the same side as the tumour.

(*b*) Incongruous contraction of isopters in the majority of cases. Incongruous bi-nasal hemianopias or bitemporal hemianopia may be found.

(c) Central scotomata are frequent generally on same side as the tumour. They are bilateral when the tumour is situated at the basal medial region.

FRANCIS J. DAMATO.

NOTES

Appointment

MR. G. HOWARD BUCK, D.O.M.S.Eng., has been appointed ophthalmic surgeon to the East Suffolk and Ipswich Hospital.

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Corrigendum

IN Mr. John Foster's letter on Lateral Orbitotomy, p. 311, line 20 should read operations in 4th, 5th and 6th case . . . in place of "operation in 4,506 cases.